An Observational study on "Clinical and Trichoscopic Patterns of Hair Loss in Systemic Lupus Erythematosus and its Correlation with Systemic Lupus Erythematosus Disease Activity Index (SLEDAI)"

A dissertation submitted in the partial fulfillment of the rules and regulations for MD DVL examination of the Tamil Nadu Dr. M.G.R Medical University, Chennai, to be held in May 2018

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DECLARATION

This is to declare that this dissertation titled — An Observational study on "Clinical and Trichoscopic Patterns of Hair Loss in Systemic Lupus Erythematosus and its Correlation with Systemic Lupus Erythematosus Disease Activity Index (SLEDAI)" is my original work done in partial fulfillment of rules and regulations for MD DVL examination of the Tamil Nadu Dr. M.G.R Medical University, Chennai to be held in May 2018.

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CERTIFICATE

This is to certify that the dissertation entitled – An Observational study on "Clinical and Trichoscopic Patterns of Hair Loss in Systemic Lupus Erythematosus and its Correlation with Systemic Lupus Erythematosus Disease Activity Index (SLEDAI)" is a bonafide work done by Dr. Shivani Bhardwaj towards the partial fulfillment of rules and regulations for MD DVL degree examination of the Tamil Nadu Dr. M.G.R Medical University, to be conducted in May 2018.

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CERTIFICATE

This is to certify that the dissertation entitled – An Observational study on "Clinical and Trichoscopic Patterns of Hair Loss in Systemic Lupus Erythematosus and its Correlation with Systemic Lupus Erythematosus Disease Activity Index (SLEDAI)" is a bonafide work done by Dr. Shivani Bhardwaj towards the partial fulfillment of rules and regulations for MD DVL degree examination of the Tamil Nadu Dr. M.G.R Medical University, to be conducted in May 2018.

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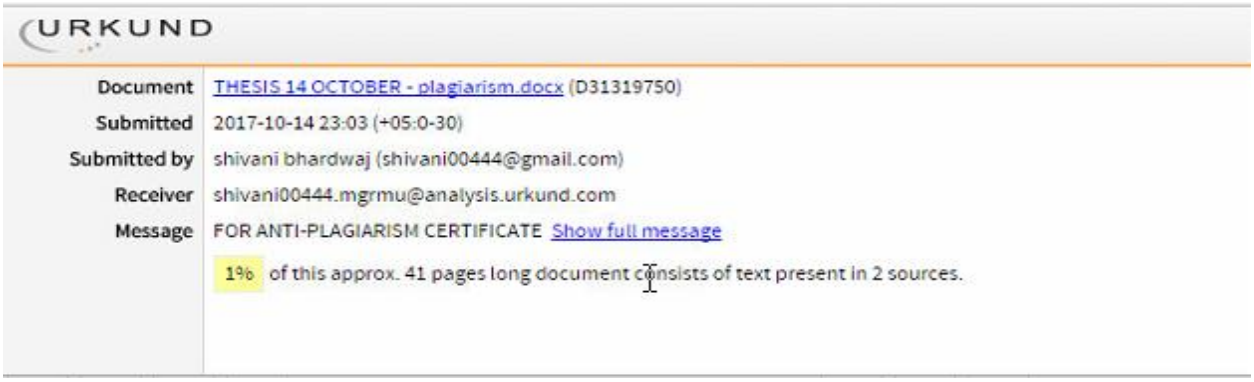
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Christian Medical College, Vellore

Dedicated to my parents

Mr. Umesh Bhardwaj

And

Dr. Ragini Sharma

ACKNOWLEDGEMENTS

This dissertation would be incomplete without expressing my gratitude to the people involved in its conceptualisation and completion.

First and foremost, I would like to thank God Almighty for giving me the opportunity and strength to pursue this topic from start to completion.

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Finally, I would like to thank *Mr. Shivang Airi* who helped to organise my work, and to lend me the grit to complete my research on time.

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Clinical and Trichoscopic patterns of hair loss in Systemic Lupus Erythematosus and its correlation with Systemic Lupus Erythematosus Disease Activity Index (SLEDAI).
Dr. Shivani Bhardwaj, Emp. No: 21117, PG Registrar, Dr. Dincy Peter, Emp. No: 31034, Professor, Dept. of DVL, Dr. Leni George, Emp. NO: 20257, Dr. Anu Anna George, Emp. No: 28939, Asst. Prof, Dr. Anjana Joseph, Emp. No: 31733, Asst. Prof, Dr. Mahabal Gauri Dinesh, Emp. No: 33506, Asst. Prof, Dr. Ramya J, Emp. No: 33219, Asst. Prof, Dr. Debashish Danda, Emp. No: 10512, Professor and Head, Dept. of Rheumatology, Ms. Reka K, Senior Demonstrator, Emp. No: 32547, Dept. of Biostatistics.

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Dear Dr. Shivani Bhardwaj,

I enclose the following documents:-

1. Institutional Review Board approval 2. Agreement

Could you please sign the agreement and send it to Dr. Biju George, Addl. Vice Principal (Research), so that the grant money can be released.

With best wishes,

Dr. Biju George
Secretary (Ethics Committee)
Institutional Review Board

Dr. BIJU GEORGE
MBBS., MD., DM.
SECRETARY - (ETHICS COMMITTEE)
Institutional Review Board,
Christian Medical College, Vellore - 632 002.

Cc: Dr. Dincy Peter, Dept. of Dermatology, CMC, Vellore

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Dear Dr. Shivani Bhardwaj,

The Institutional Review Board (Blue, Research and Ethics Committee) of the Christian Medical College, Vellore, reviewed and discussed your project titled "Clinical and Trichoscopic patterns of hair loss in Systemic Lupus Erythematosus and its correlation with Systemic Lupus Erythematosus Disease Activity Index (SLEDAI)" on September 05th 2016.

The Committee reviewed the following documents:

1. IRB Application format
2. Consent Forms and Patient Information Sheets(English, Tamil, Telugu, Hindi, Malayalam)
3. Cvs of Drs. Anjana, Anu Anna George, Dabasish Danda, Dincy Peter, Gauri, Lini George, Ramya J.
4. Signature Pages
5. No. of documents 1 – 4

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Additional Vice-Principal (Research)

The following Institutional Review Board (Blue, Research & Ethics Committee) members were present at the meeting held on September 05th 2016 in the C K Job Hall, Paul Brand Building, Christian Medical College, Bagayam, Vellore 632002.

Name	Qualification	Designation	Affiliation
Dr. Biju George	MBBS, MD, DM	Professor, Haematology, Research), Additional Vice Principal , Deputy Chairperson (Research Committee), Member Secretary (Ethics Committee), IRB, CMC,Vellore	Internal, Clinician
Dr. B. J. Prashantham	MA(Counseling Psychology), MA (Theology), Dr. Min (Clinical Counselling)	Chairperson, Ethics Committee, IRB. Director, Christian Counseling Centre, Vellore	External, Social Scientist
Dr. Anuradha Rose	MBBS, MD, MHSC (Bioethics)	Associate Professor, Community Health, CMC, Vellore	Internal, Clinician
Dr. Jayaprakash Muliyil	BSc, MBBS, MD, MPH, Dr PH (Epid), DMHC	Retired Professor, Vellore	External, Scientist &Epidemiologist
Rev. Joseph Devaraj	BSc, BD	Chaplaincy Department, CMC, Vellore	Internal, Social Scientist
Mr. C. Sampath	BSc, BL	Advocate, Vellore	External, Legal Expert
Dr. Visalakshi. J	MPH, PhD	Lecturer, Biostatistics, CMC, Vellore	Internal, Statistician
Mrs. Sheela Durai	MSc Nursing	Professor, Medical Surgical Nursing, CMC, Vellore	Internal, Nurse
Ms. Grace Rebekha	M.Sc., (Biostatistics)	Lecturer, Biostatistics, CMC, Vellore	Internal, Statistician
Mrs. Pattabiraman	BSc, DSSA	Social Worker, Vellore	External, Lay Person
Dr. Rajesh Kannangai	MD, PhD.	Professor, Clinical Virology, CMC, Vellore	Internal, Clinician

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Dr. Sathish	MBBS, MD, DCH	Professor, Child Health, CMC, Vellore	Internal, Clinician
Dr. Inian Samarasam	MS, FRCS, FRACS	Professor, Surgery, CMC, Vellore	Internal, Clinician
Dr. Mathew Joseph	MBBS, MCH	Professor, Neurosurgery, CMC, Vellore	Internal, Clinician
Dr. Ranjith K Moorthy	MBBS, MCh	Professor, Neurological Sciences, CMC, Vellore	Internal, Clinician

We approve the project to be conducted as presented.

Kindly provide the total number of patients enrolled in your study and the total number of withdrawals for the study entitled: "Clinical and Trichoscopic patterns of hair loss in Systemic Lupus Erythematosus and its correlation with Systemic Lupus Erythematosus Disease Activity Index (SLEDAI)" on a monthly basis. Please send copies of this to the Research Office (research@cmcvellore.ac.in).

Fluid Grant Allocation:

A sum of 50,000/- INR (Rupees Fifty Thousand Only) will be granted for 12 Months.

Yours sincerely,

Dr. Biju George
Secretary (Ethics Committee)
Institutional Review Board

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Christian Medical College, Vellore - 632 002.

IRB Min No: 10266 [OBSERVE] dated 05.09.2016

CHRISTIAN MEDICAL COLLEGE, VELLORE
AGREEMENT TO BE SIGNED BEFORE RELEASE OF ANY RESEARCH GRANT
*****

1. I understand that the research grant is sanctioned only for the specific project approved by the Institutional Review Board and should be used exclusively for this project

2. I note that the project will become operational with effect from the date on which the grant is received, and I agree to complete it within the stipulated time of 12 months.

3. I agree to submit promptly and regularly, the periodical (Half Yearly for One Year Project/Annually for Two years project) reports and the final report of the work done, in the approved format.

4. If I plan to leave the institution on before the completion of the project. I will submit a complete and detailed report of the work done by me on the project till the date of relief and transfer the project, either to the Guide or to the Co-Investigator for completion and submission of the Final Report.

5. I agree that any publication arising out of this project will carry an acknowledgement of the financial support of the Christian Medical College Fluid Research Fund.

Fluid Grant Allocation:

A sum of 50,000/- INR (Rupees Fifty Thousand Only) will be granted for 12 Months.

PRINCIPAL

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Project Title: Clinical and Trichoscopic patterns of hair loss in Systemic Lupus Erythematosus and its correlation with Systemic Lupus Erythematosus Disease Activity Index (SLEDAI).

Ref: IRB Min No: 10266 [OBSERVE] dated 05.09.2016

ABBREVIATION

AA	Alopecia areata
ACR	American College of Rheumatology
AGA	Androgenetic alopecia
ANA	Antinuclear antibody
ARA	American Rheumatism Association
BILAG	British Isles Lupus Assessment Group
CLASI	Cutaneous Lupus Erythematosus Disease Area and Severity Index
DCS	Dissecting cellulitis of scalp
DLE	Discoid lupus erythematosus
DNA	Deoxyribonucleic acid
FAGA	Female androgenetic alopecia
HCP	Honeycomb pigment
HDD	Hair diameter diversity
IRS	Inner root sheath
IQR	Interquartile range
LE	Lupus erythematosus
LPP	Lichen planopilaris
NSAIDS	Non-steroidal Anti-inflammatory Drugs
ORS	Outer root sheath
SCLE	Subacute cutaneous lupus erythematosus
SFU	Single follicular units
SLAM	Systemic Lupus Activity Measure
SLE	Systemic lupus erythematosus
SLEDAI	Systemic lupus erythematosus disease activity index
SLICC	Systemic Lupus International Collaborating Clinics criteria
TE	Telogen effluvium
TSH	Thyroid stimulating hormone

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INTRODUCTION

Systemic lupus erythematosus (SLE) is a multisystem connective tissue disease with considerable mortality and morbidity. Systemic lupus erythematosus is associated with immunological abnormalities. It has a striking diversity of clinical patterns, pathogenesis and prognoses. They usually present with clinical features including fever, photosensitive skin rashes and arthritis. Renal involvement is common but pulmonary, cardiac and neurological involvement may also occur. The diagnostic criteria for SLE was developed by the American Rheumatism Association (ARA) in 1971 which was later modified in 1982 (1). Skin manifestations are one of the most common presenting symptoms in a patient with SLE. They are important in diagnosing SLE: cutaneous features account for four of the 11 revised American College of Rheumatology (ACR) criteria for the classification of SLE (2). The Systemic Lupus International Collaborating Clinics (SLICC) criteria introduced in 2012, included 17 criteria and there were four cutaneous features among the 11 clinical criteria. In comparison to ACR criteria 1982, Systemic Lupus International Collaborating Clinics (SLICC) criteria has greater sensitivity but lower specificity for the diagnosis of SLE (3).

Alopecia in SLE is common and is present in 20-60% of the patients (4). The hair loss in SLE may be correlated with the disease activity index (5). Hair loss in SLE can be either scarring or non-scarring. Diffuse non-scarring alopecia is the most common non-specific skin manifestation of SLE, and occurs in more than 60% of cases, either as transiently or during increased disease activity. Alternatively, the alopecia can be chronic, which can

lead to coarse, dry, and fragile hair along the peripheral hairline during a systemic exacerbation and are known as ‘lupus hair’. Alopecia areata is also more often reported, in approximately 10% of patients with SLE. Discoid lupus erythematosus is a specific cutaneous manifestation of SLE, and it results in permanent scarring alopecia. (6–8). Alopecia can cause considerable psychological impact as it causes intense emotional suffering, and leads to personal, social, and work related problems (9).

Measurement of disease activity is necessary for evaluation and management of SLE. Systemic lupus erythematosus disease activity index (SLEDAI) scoring is a global scoring system which was introduced in 1985, and was modified in 2002. It comprises of twenty four items for the nine organs/systems. Score range from 0–105 points. Alopecia has a score of one point in this scoring system (10–12).

In this study, we have described the various patterns of alopecia seen in patients with SLE and correlated with disease activity (SLEDAI). The trichoscopic patterns seen in these patients were also described.

AIMS AND OBJECTIVES

1. To observe the clinical and trichoscopic patterns of different types of alopecia seen in systemic lupus erythematosus.

2. To study the patterns of hair loss in systemic lupus erythematosus and its correlation with SLE disease activity index (SLEDAI score).

REVIEW OF LITERATURE

1. INTRODUCTION

Lupus erythematosus (LE) is a term designated to a group of illnesses with an underlying abnormality of autoimmunity directed towards the molecular constituents of nucleosomes and ribonucleproteins (13). Lupus can manifest in a spectrum, from a mild disease with only skin features as discoid LE, to a range of systemic features as in systemic LE (14).

2. HISTORY

Ferdinand von Hebra first described the skin lesions with aggressive and tissue destructive nature in 1845 (15). Pierre Louis Cazanave coined the term "lupus erythemateaux " in 1851 due to its appearance to wolf bite (15,16). In 1872, Kaposi reported certain patients suffering from lupus presented with a syndrome consisting of fever, arthritis, lymphadenopathy, and anaemia. This report separated systemic lupus erythematosus (SLE) from the other forms of cutaneous LE (15). Klemperer *et.al.* gave the hypothesis of SLE as a connective tissue disorder (15). In 1901, Paul Ehrlich described the concept of autoimmunity and with this the era of immunology began (16). In 1910, Hauck reported a high incidence of false-positive results in the Wassermann reagin test in lupus patients. Hargraves, described the "LE cell phenomenon" in 1948 followed by the introduction of the LE cell test by Haserick *et.al.* in 1949. Holman *et.al.* presented evidence that the LE cell factor was an antibody reacting with

deoxyribonucleic acid, and these reports led to the clinical use of antinuclear antibody (ANA) assays in routine diagnostic measures in the late 1950s (15).

3. EPIDEMIOLOGY

The prevalence rate in a comparative analysis by Danchenko *et.al.* has been reported to be 52/100,000 population in United States with higher rates among Hispanic and black group (17). The prevalence rate of disease in Asia ranged from 30 to 50/100,000 population (18). In a study by Feldman *et.al.* in 2013, the overall prevalence of SLE was 143.7/100,000 in U.S and was 6 times more in females than in males. The incidence rate of SLE was 23.17 per 100,000 person-years in this study (19).

A prevalence study in India (carried out in a rural population near Delhi) found a point prevalence of 3 per 100,000, which is much lower than the western population (20). However, larger epidemiological studies are needed to confirm the finding of these studies regarding the prevalence of this disease in India. The reported age of onset of disease varied from 24.5 years seen in the study conducted by Malaviya *et.al.* in 1997 from India (21) to a much later age of onset, as seen in black people and white people where it was 39.4 ±15.9 years, and 45.4 ± 17.7 years respectively (22). The sex ratio seen in Feldman *et.al.* in 2000-2004 was 6:1 (F: M) (19), though this ratio was much lower than the study by Malaviya *et.al.* (21, 23). The age of onset and sex ratio in various studies is shown in Table 1.

***Table 1:** The age of onset and sex ratio in various studies(NA-Data not available)*

Parameter	**Feldman *et.al.*** (19) **n = 34,339** **2000-2004**	**Garris *et.al.*** (24) **n = 13,348** **2003-2007**	**Sadana *et.al.*** (25) **n = 20** **1955-1963**	**Soto *et.al.*** (26) **n = 187** **1982-2002**	**Jarukitsopa *et.al.*** (27) **n = 45** **1993-2005**	**Yun *et.al.*** (28) **n = 122** **2004**	**Malviya *et.al.*** (21) **n=1366** **1997**
Type of study	Retrospective	Retrospective	Retrospective + prospective	Retrospective	Retrospective	Crossectional study	Retrospective
Ethnicity	Low-income U.S. Medicaid population	US Medicare population	Indian	Mexico	White US population	Korean	Indian
Mean age and Age range (years)	Mean age- NA Age range - 18 - 65	Mean age -61 Age range – NA	Mean age- NA Age of onset range- 10-53	Mean age- 31 Age range- F- 10-75 M- 7-65	Mean age- 42 Age range- NA	Mean age- 32.7 Age range- 13-71	Mean age- 24.5 Age range- 4-75
Female: Male Ratio	6:1	6:1	13:7	4.2:1	10:1	12.6:1	11:1

4. DIAGNOSTIC CRITERIA FOR SLE

In 1971, American college of rheumatology introduced 14 criteria comprising of facial erythema, discoid lupus, Raynaud's phenomenon, alopecia, photosensitivity, oral or nasal ulcers, arthritis without deformity, LE cells (2 or more), chronic false positive serology for syphilis (more than 6 months), proteinuria, cellular casts, pleuritis or pericarditis, psychosis or convulsions, haemolytic anaemia or leucopenia or thrombocytopenia (29) for the diagnosis of SLE (Annexure 1). The presence of 4 or more of the above criteria is considered as diagnostic of SLE.

In 1982, ACR revised the above criteria and the modified ACR criteria was introduced which comprised of 11 criteria. Alopecia was not included in this revised classification for SLE. The presence of 4 or more of the 11 criteria, serially or simultaneously, during any interval or observation is considered as diagnostic of SLE (30) (Annexure 2).

In 2012 Petri *et.al.*, proposed SLICC criteria (Systemic Lupus International Collaborating Clinics classification criteria for systemic lupus erythematosus) for the SLE diagnosis. According to the SLICC criteria, the patient must satisfy at least 4 criteria, including at least one clinical criterion and one immunologic criterion OR the patient must have biopsy-proven lupus nephritis in the presence of antinuclear antibodies or anti-double-stranded DNA antibodies (31) (Annexure 3). Sensitivity and specificity of SLICC criteria was found to be 94% and 92% respectively in comparison to the ARA criteria which was 86% and 93% respectively. Thus, SLICC criteria was more sensitive though less specific than ARA criteria. Non-scarring alopecia was introduced as a major clinical criterion in

the SLICC diagnostic criteria. Diffuse thinning or hair fragility with visible broken hairs, in the absence of other causes such as alopecia areata, drugs, iron deficiency, and androgenic alopecia were included under this criterion.

5. CUTANEOUS LUPUS ERYTHEMATOSUS

Cutaneous manifestations can be divided into (13)

- *LE specific lesions* which show interface dermatitis on histopathology.
- *LE- nonspecific lesions* which are not specifically seen in LE and/ or may be seen in other skin diseases and do not show distinct LE histopathology.

Lupus erythematosus specific skin disease is also termed as cutaneous LE which can be further divided into acute cutaneous LE, subacute cutaneous LE, and chronic cutaneous LE. Lupus erythematosus specific and lupus erythematosus non-specific skin lesions are further classified by Gilliam in 1981(8) (Annexure 4)

Cutaneous features may provide an insight to the position of the patient, on the spectrum of lupus erythematosus, as the patient with acute cutaneous LE have greater chances of having haematological and serological features in comparison to chronic cutaneous LE (13). Though LE non-specific skin lesions do not enable a diagnosis of LE on their own, they can be important in reflecting the underlying SLE disease activity. The most common symptoms seen in SLE is arthralgia followed by mucocutaneous lesions (6). The predominant mucocutaneous symptoms of SLE shown in various studies is listed in Table 2.

Table 2: Mucocutaneous symptoms in SLE in various studies

Clinical finding	Malaviya *et.al.* 1988 (32)	Malaviya *et.al.* 1997 (21)	Kosaraju *et.al.* 2010 (33)	Saigal *et.al.* 2011 (34)	Kapadia *et.al.* 1996 (35)	Agarwal *et.al.* 2013 (36)	Pankaj *et.al.* 2011 (37)
Malar rash	85	58.5	35.41	43.3	60	71.3	31.2
Discoid rash	NA	7	NA	1.7	57.5	32.2	54.8
Alopecia	**82**	**71**	**18.75**	**65**	**82.5**	**10.34**	**64.5**
Photosensitivity	67	48	27.08	75	60	63.2	NA
Oral ulcers	64	57	25	61.7	60	42.53	41.9

6. *PATHOPHYSIOLOGY OF ALOPECIA IN LUPUS ERYTHEMATOSUS*

6.1 Anatomy of hair and hair cycle:

Hair is derived from epidermis. Hair has two separate structures, the follicle in the skin and the hair shaft, externally on the body surface. On the outer surface it is a fully keratinized epithelial cells. The inner portion is a part of individual living hair follicular unit with cylindrical epithelial down growths into the dermis and subcutaneous fat, where it enlarges at the base into the hair bulb which is surrounded by mesenchymal derivative called dermal papilla (38). (Figure 1)

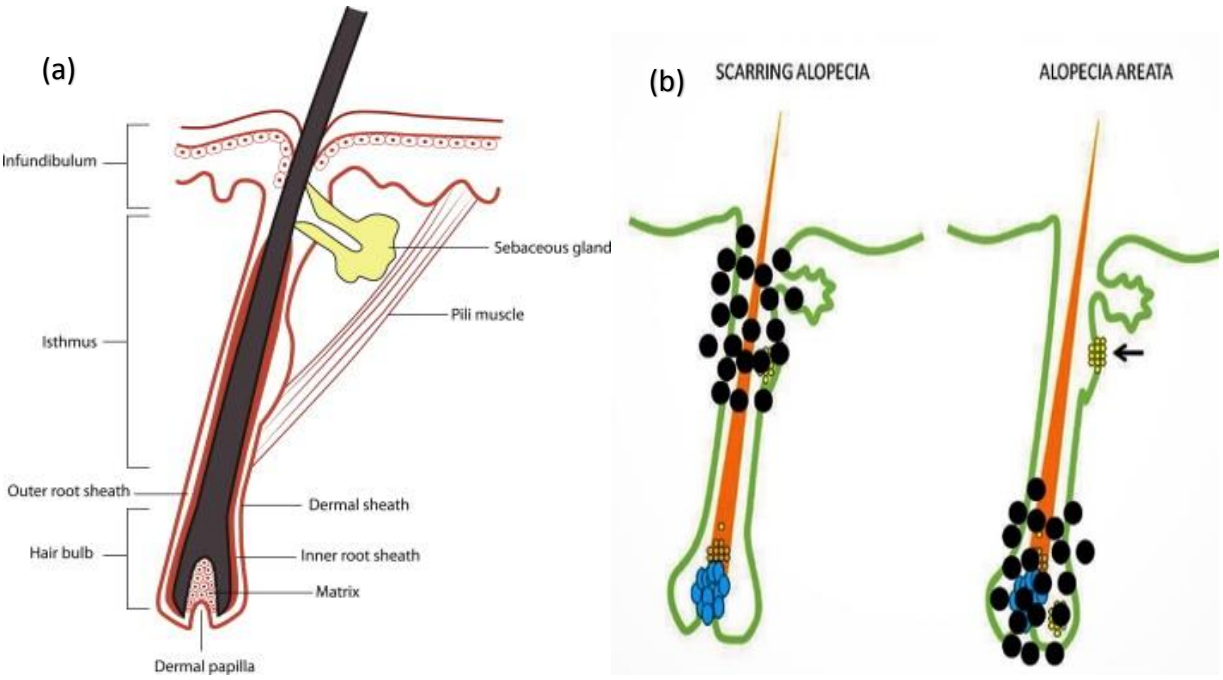


Figure 1: (A) Anatomy of hair (B) Showing site of inflammation in scarring and non-scarring (e.g. alopecia areata) alopecia.

The hair shaft consists of a medulla in the central region, cortex which surrounds the medulla and is covered by the cuticle. The follicle is the essential growth structure of

hair. It is composed of the outer root sheath (ORS) and an inner root sheath. The outer root sheath has a reservoir of multipotent stem cells, i.e. keratinocyte and melanocyte stem cells and also contains keratinocytes. It forms a distinct bulge area between the insertion of the arrector pili muscle and duct of the sebaceous gland. The inner root sheath (IRS) consists of three layers: Henle's layer, Huxley's layer, and cuticle layer. The IRS cuticle lies adjacent to the cuticle of the hair shaft, anchoring the hair shaft to the follicle (38).

The hair is produced by a portion of the follicle called hair bulb. The hair bulb encloses the follicular dermal papilla, dermal papilla cells, mucopolysaccharide-rich stroma, nerve fibers, and a single capillary loop. The follicular papilla plays an important role in hair growth and essential for the growth factors like bone morphogenetic protein, hepatocyte growth factor, insulin-like growth factor, stem cell factor which are critical for hair growth and melanogenesis. The hair bulb is formed of two regions: a lower region of undifferentiated cells and an upper region in which the cells became differentiated. The Auber's line across the widest part of the papilla separates the two regions. Below this lies the matrix or germination center of the follicle and the dermal papilla (38).

Above the hair bulb, the upper hair follicle is divided into two anatomical parts: the infundibulum and the isthmus. The infundibulum is a funnel-shaped structure filled with sebum and it extends from the surface of the skin to the sebaceous duct. In the acroinfundibulum which is the upper part, the epithelium is continuous with the keratinized epidermis and is covered by an impermeable stratum corneum. In

infrainfundibulum which is the lower follicular portion this is interrupted as the differentiation pattern switches from epidermal differentiation to a tricholemmal differentiation pattern. The isthmus extends from the duct of the sebaceous gland to the insertion of arrector pili muscle (38).

The hair growth happens in repeated cycles which can be divided into three distinct phases: (a) Anagen or growth phase (b) Catagen or transitional phase and (c) Telogen or resting phase. The stages of rapid growth and hair shaft formation alternate with stages of apoptosis-driven hair follicle regression.

In each hair cycle, remodeling of the temporary lower hair follicle which is below the isthmus or site of attachment of the arrector pili, occurs. The epidermal and dermal follicular cell populations that reside in the upper permanent portion of the hair follicle impart this regenerative capacity. The reservoir of slow-cycling, pluripotent cells exists near the bulge which is a portion of ORS where arrector pili muscle attaches. These pluripotent stem cells produce secondary germ cells that migrate bidirectionally and undergoes coordinated differentiation to regrow the lower hair follicle during normal telogen-anagen cycling, and to restore and renew the upper follicle, including the sebaceous gland, and adjacent epidermis. The follicular dermal elements encompasses the dermal papilla and dermal sheath, both of which approximate the bulge area during telogen. They have a prime inductive and regulatory roles with onset of anagen.

Langerhans cells, which are concentrated in the infundibular epithelium, bulge, and sebaceous epithelium, probably initiate a first-line immune response to exogenous and endogenous antigenic threats to follicular viability (39).

6.2 Pathophysiology of scarring alopecia in SLE:

Typical LE- specific cutaneous lesion of disease on the scalp is discoid lupus erythematosus (DLE). In general, 60% of DLE patients can have scalp involvement but isolated scalp involvement is seen in 10% of patients. Discoid lupus erythematosus lesions can be seen in 15-30% of patients with SLE (13). In view of the high specificity of the discoid lesion, it has been included in the criteria for the classification of SLE. Five to ten percentage of patients presenting with DLE lesions may have SLE. The extent and distribution of the DLE lesions determine the risk of development of SLE. Patients with disseminated DLE (lesions both above and below the neck) have a higher rate of immunological abnormalities and risk for progression to SLE in comparison to the patients with localised DLE (lesions restricted to the head and neck area) (40). A DLE lesion over scalp begins as a well-demarcated round or oval purplish macule or papule and enlarges into an alopecic patch with follicular plugging, erythema, and adherent scaling. The lesions may be hypopigmented or hyperpigmented. They may present with itching, pain, burning or tenderness. End stage disease is characterized by atrophic, fibrotic, smooth white plaques with loss of follicular ostia (41). The most common site observed by Thakur *et.al.* in 2015 was occipital scalp in females and frontoparietal scalp in males (42). Discoid lupus erythematosus has been classified as primary lymphocytic

cicatricial alopecia. The scarring or cicatricial alopecia results from irreversible damage to the epithelial hair follicle stem cells which are present in the bulge (Figure 1). The inflammation in the area of the bulge destroys the potential for regeneration of the hair follicle by causing destruction of the epithelial hair follicle stem cells (43). In 2008, the study by Al-Refu *et.al.* (44) demonstrated the cytokeratin 15 (CK 18) staining by C8 /144B antibody (antibody to the CD8 antigen) on hair follicle stem cell. They reported that with mild to moderate inflammation, the staining showed normal to moderate CK15 expression at the bulge region where as in severe inflammation, the expression of CK15 was weak or absent.

A combination of genetic, environmental, and host factors determines the pathogenesis of DLE. It is thought that in susceptible individuals, ultraviolet light exposure plays a key role inducing apoptosis of keratinocytes and a reactive T-cell- or immune-complex-mediated response to cause the disease (39). The reduction of Bcl-2 expression in the basal cells is associated with the overexpression of Fas antigen and correlates directly with the extent of apoptosis in the epidermis (45,46). Another important trigger for DLE is thought to be the Koebnerization. Discoid lupus erythematosus is known to occur in areas of excoriation and trauma (39). On histology, the inflammation is typically more around the mid follicular level where sebaceous glands are present. There is an early histologic damage to the sebaceous glands in the form of lymphocytic infiltration of the sebaceous glands and the disruption of glandular structure which leads the sebaceous glands be the first target in DLE and they are the first adnexal structures to disappear even before the hair follicles in DLE. (47–49).

6.3 Pathogenesis of non-scarring alopecia in SLE

The non- scarring alopecia in SLE can be-

1. Diffuse non-scarring alopecia –
    a) Telogen effluvium
    b) Androgenetic alopecia
    c) Anagen effluvium
    d) Lupus hair
2. Patchy non-scarring alopecia – Alopecia areata

- *1.(a) Telogen effluvium-*

Systemic lupus erythematosus patients can have a diffuse, non-scarring, and transient hair loss which is usually associated with exacerbations of the disease process. This diffuse hair loss in SLE is usually the result of a telogen effluvium. It is a result of both severe catabolic effects and an effect of elevated levels of circulating proinflammatory cytokines of the lupus disease flare on hair growth cycling (40).

The term telogen effluvium, introduced by Kligman in 1961 (50), refers to abrupt generalised shedding of telogen hairs. It can be acute, when duration of hair loss is less than 3 months, or chronic, when duration of hair loss is more than 3 months. It may be triggered by intrinsic or extrinsic factors that cause a large number of hairs to enter in the telogen phase at one time. These hairs shed about 3–4 months after exposure to the triggering factor. These factors include acute febrile illness, psychological stress,

pregnancy, thyroid diseases, crash diets, iron deficiency, discontinuation of oestrogen-containing drugs, medications (beta-blockers, anticoagulants, retinoids, propylthiouracil, carbamazepine, and vaccines), major surgery and ultraviolet exposure. This is the most common type of alopecia found in the systemic lupus erythematosus.

The functional classification of telogen effluvium given by Ralph *et.al.* in 2016 is as follows:

- Immediate anagen release- the follicles prematurely enter telogen that would normally complete a longer cycle by remaining in anagen. This is a common form of TE, occurring after periods of physiologic stress.
- Delayed anagen release- hair follicles remain in prolonged anagen rather than cycling into telogen. And when they are finally released from anagen, the clinical sign of increased shedding of telogen hair will be found.
- Immediate telogen release- the hair follicles normally programmed for release of telogen hairs after an interval of usually 100 days after the end of anagen are prematurely stimulated to cycle into anagen. There is premature teloptosis.
- Delayed telogen release- the hair follicles remain in prolonged telogen rather than being shed and recycling into anagen; when finally teloptosis sets in, again the clinical sign of increased shedding of telogen hair is observed.
- Short anagen phase- it results in a mild form of persistent telogen effluvium in association with decreased hair length.

- *1. (b). Androgenetic alopecia (AGA)*

Androgenetic alopecia is an androgen-related condition that develops in genetically predisposed individuals. Male pattern hair loss (male AGA) and female pattern hair loss (female AGA) share a similar pathogenic pathway and the same histopathology of hair follicle miniaturization. Dihydrotestosterone binds to the androgen receptors in the susceptible hair follicles and the hormone-receptor complex activates the genes which gradually transform large terminal follicles to miniaturized follicles (51–54). Jang *et.al.* in 2013 (55), retrospectively studied in Korean patients the age of onset and severity of AGA in both genders, and reported that the mean age of onset of AGA in 2010 was 31.6 years. This study also compared the mean age of onset of AGA from 2006 to 2010 and found that the age of onset of AGA in 2010 is earlier (31.6 years) in comparison to 2006 (34.1 years). In 2016, Erdogan *et.al.* postulated that there is an increased oxidative stress in patients with early-onset AGA (56).

- *1. (c). Anagen effluvium-*

It is a term for multiple conditions associated with diffuse hair loss from follicles in the anagen growth phase. It can occur due to cytotoxic drug or other toxic factors, non-cytotoxic agents (e.g., acitretin), exposure to radiation therapy or toxins, and from systemic disorders (41). This type of diffuse non-scarring alopecia can occur in SLE with severe systemic disease as dystrophic anagen effluvium. In this, episodes of severe illness result in a temporary shutdown of the hair matrix, producing a narrowed segment of hair shaft (Pohl–Pinkus constriction), which are prone to intrafollicular fractures (40).

- *1.(d) Lupus Hair-*

The *Lupus hair* is usually dry, coarse and fragile hair especially on frontal margin and the periphery of the scalp causing receding frontal hairline where hairs are broken due to hair follicle growth retardation. Lupus hair is seen as transient alopecia in chronically active SLE patients which closely relate to telogen effluvium and causes thin, weakened hairs or lupus hairs, especially at the periphery of the scalp. This leads to an unruly appearance with short, broken-off short hair, which do not get combed easily and produce a disheveled appearance (57,58). There is a hypothesis that the normal hair growth is interrupted with the induction of a negative nitrogen balance and leads to the production of thin, weakened hairs which easily fragment above the surface of the scalp (40).
The pathomechanisms which leads to generalized hair loss in SLE do not result in scarring. With the remission of the disease activity, the hair regrowth is expected and the alopecia disappears (40).

- *2. Alopecia areata –*

The patchy non-scarring alopecia in SLE is most commonly the alopecia areata. It is a common disease that results in the loss of hair on the scalp and elsewhere on the body. There are three types of alopecia areata based on extent of involvement; patchy alopecia areata, alopecia totalis and alopecia universalis (59). Based on the pattern of involvement it could be reticular, ophiasis or sisaipho type. A new variant described is acute and diffuse total alopecia mainly seen among females. Other unusual patterns are perinevoid alopecia and linear alopecia areata (60). In a study by Werth *et.al.* in 1992, alopecia

areata was found in 10% of the patients in the cohort of 39 patients in comparison to 0.42% in general dermatologic patients (61). On histopathology, there is a peribulbar inflammation and therefore, the follicles are not permanently destroyed because the bulge region is unaffected by the disease process (43). Occasionally another form of patchy hair loss which occurs in patients with severe disease in which there are patches of partial hair loss scattered on the scalp, associated with mild erythema but no evidence of scarring. Complete hair regrowth occurs with disease remission. A peribulbar infiltrate of lymphoid cells is found surrounding anagen hair bulbs which is denser than that found in alopecia areata (40).

- *3.* Hair loss can also manifest as an *adverse effect of therapeutic management* of SLE like Non-steroidal Anti-inflammatory Drugs (NSAIDS), mycophenonate mofetil, methotrexate, cyclophosphamide and acitretin (62–65).

Alopecia is observed in SLE patients ranging from 20-60% as studied in a Korean population (4). More commonly event of diffuse non-scarring hair loss happens at the onset which may be one of the first symptoms of the disease and the hair grow back when the disease is under control (66). Patients with hair loss have been found with higher rate of cutaneous manifestations, Raynaud's phenomenon and muscle pain. Most of these signs correlate with the severity of alopecia. In a study of Wysenbeek AJ *et.al.* alopecia correlated with disease activity index (5). As described by Sook Jung YUN *et.al.* from Korea in 2007, non-scarring pattern can be diffuse or patchy and identified as telogen effluvium (65.1%), female pattern hair loss (10.5%), anagen effluvium (12.8%), 'lupus hair' (15.1%) and alopecia areata (15.1%). Patients with SLE can have scarring alopecia

as seen with discoid lupus erythematosus (7%) (4,67). The study in SLE patients conducted by P Salphale *et.al.* (37) in 2011 from south India showed non-scarring alopecia in 64.5% patients, telogen effluvium in 73.3% patients, lupus hair in 25% patients and alopecia areata in 1.7% patients. Kapadia *et.al.* in 1996 noticed non-cicatricial diffuse alopecia in (82.5%), cicatricial alopecia in (15%), and lupus hair (12.5%) among 40 patients with SLE (35). The types of alopecia in SLE in various studies are shown in Table 3.

Table 3: Types of alopecia in SLE in various studies.

Type of alopecia	**Yun *et.al.* (7)**	**P Salphale *et.al.*** (37)	**Kapadia *et.al.*** (35)
Telogen effluvium	65.1%	73.1%	Diffuse nonscarring alopecia- 82.5%
Androgenetic alopecia	10.5%	NA	-NS
Anagen effluvium	12.8%	NA	-NS
Lupus hair	15.1%	25%	12.5%
Alopecia areata	15.1%	1.7%	NA
DLE	7%	16.1%	15%

NA- data not available, NS- not specified separately

7. TRICHOSCOPY

The term “Trichoscopy”, coined by Rudnicka and Olszewska in 2006, refers to the evaluation of hair and scalp using a dermoscope (68). Dermatoscopy or dermoscopy also known as surface microscopy or epiluminescence microscopy, is a non-invasive technique which allows easy, quick and magnified observation of the morphological features of the skin which are imperceptible to the naked eyes (69). Both dermoscopy and trichoscopy can be performed with manual devices which generally employs X 10 magnification. Videodermoscopy, is an evolved dermoscopy performed with a video-camera with magnifying lenses with magnification range of X 10 to X 1000. The images are visualized on a computer screen. Images can be stored and can be compared with previous or future images of the same patient. This is performed by an epiluminescence microscopy technique with an aid of a liquid (water, alcohol or oil) to the skin to eliminate the light reflection (69).

Hair shaft thickness of the normal scalp

In 2009, a study conducted by Rakowska *et.al.* in Polish population (70) showed the mean thickness of hair as 0.061 mm in the frontal scalp and 0.058 mm in the occipital scalp. Temporal scalp hair thickness was between 0.058-0.061 mm. It was described that the hair can be thin (<0.03mm), medium-sized (0.03-0.05mm) or thick (>0.05mm) in size. The mean proportion of thin, medium and thick sized hair is approximately 6, 21 and 73% respectively. Therefore, limited hair shaft diameter heterogeneity is normal (71). The trichoscopic patterns seen by dermoscope can be classified as shown in Table 4 (72).

Table 4: Trichoscopic patterns seen by dermoscope

Follicular patterns	*Inter-follicular pattern*	*Hair shaft patterns*	*Hair roots through the scalp*
1. Yellow dots 2. Pinpoint white dots 3. Red dots 4. Blue–gray dots 5. Keratotic plugs 6. Gray–white halos 7. Peripilar signs 8. Empty follicles 9. Loss of follicular openings	1. Scales -Interfollicular -Peripilar casts 2. Vessels -Simple red loops -Arborizing vessels -Twisted red loops -Giant capillaries 3. Honeycomb pigment 4. White patches	1. Hair diameter diversity (HDD) 2. Short regrowing hairs 3. Circle hairs 4. Hair tufting 5. Broken hair: -Exclamation mark hairs -Caudability hairs -Broken hairs -Monilethrix-like hair -Cadaverized hairs or“black dots” -Question mark hairs -Comma hairs -Flame hairs -Corkscrew hairs	1. Scalp atrophy due to steroids 2. Aplasia cutis congenita 3. Erosive pustulosis of the scalp

The description of relevant features pertaining to this study has been described:

Yellow dots-

Initially proposed by Ross *et.al.* (73), are marked by yellow to yellow-pink, round or polycyclic dots that vary in size and are uniform in color. They represent distention of the affected follicular infundibulum with keratinous material and sebum. In alopecia areata, degenerating follicular keratinocytes probably constitute the bulk of the yellow dots.
In a study conducted by Inui *et.al* , (74) yellow dots were seen in 63.7% of cases in contrast to Ross *et.al.* study where 94.8% cases with alopecia areata had yellow dots (73). In a study by Naveen *et.al.*2013 in South India, the yellow dots were found in 57.33% of patients (75). Similarly Ankad *et.al.* found yellow dots in 50% of patients. A study from North India by Chiramel *et.al.* found 87.5% patients of alopecia areata with yellow dots. The low incidence was attributed to the skin color of South Indian patients which might make the yellow dots difficult to perceive and also due to the hair care practices. Yellow dots in discoid lupus erythematosus are larger in size and may correspond to follicular plugging (76–78).

Pinpoint white dots-

These are seen in normal scalp and interspersed between hair follicles. They correspond to the sweat glands and follicular openings. They are increased in all types of alopecia. (70, 71).

Blue gray dots-

Blue-gray dots corresponds to the melanin incontinence. Target pattern blue-grey dots in a target pattern are seen in lichen planopilaris (LPP) and indicates the circular arrangement of melanin around the perifollicular area sparing the interfollicular area. In DLE blue-gray dots are seen in speckled pattern and indicates the involvement of interfollicular areas with sprinkling of melanin in these areas (78, 79).

Gray–white peripilar halos-

They present as 0.3–0.5 mm in diameter white-grayish circle surrounding a single hair or multifollicular ostium. They correspond to the affected follicles with the surrounding zone of lamellar perifollicular fibrosis. Most commonly they are seen in centrifugal cicatricial alopecia and discoid lupus erythematosus (71, 80).

Red dots-

Follicular red dots appear as erythematous polycyclic, concentric structures, with a diameter range of 0.16 to 0.47 mm, regularly distributed in and around the follicular ostia. Histopathologically it shows widened infundibula plugged by keratin and surrounded by dilated vessels and extravasated erythrocytes. The follicular red dot pattern is a specific feature of scalp lesions of active lupus erythematosus of the scalp (82).

Peripilar sign-

It is seen in 90% of the males and 86% of the females with androgenetic alopecia. It is a feature of early androgenetic alopecia. In 2004, Claire Deloche *et.al.* described histopathology of peripilar sign as superficial perivascular and interstitial infiltrate of

lymphocytes, few mastocytes, and sometimes dilated capillaries in the papillary dermis. There were no dermal melanocytes in or near the infundibulum (83).

Empty follicles-

These are seen in nonscarring alopecias, including androgenetic alopecia and telogen effluvium. They appear as empty follicular openings.

Hair diameter diversity (anisotrichosis) –

In 2001, Oliver de Lacharrière *et.al.* (84) described the hair diameter diversity as an important indication of hair follicle miniaturization and its importance in the diagnosis of androgenetic alopecia. Diversity in hair diameter results due to the hair follicle miniaturization in androgenetic alopecia, which does not affect all hair follicles at same time and thus results in presence of terminal, indeterminate and miniaturized hair simultaneously. In this study, hair diameter diversity more than 20 percent was considered to be suggestive of androgenetic alopecia.

Coudability hairs –

These are normal-looking long hairs tapered at the proximal end. In 1984, Shuster first decribed the coudability sign as normal-looking hair which can be easily bent or pushed inward. The appearance of the kink gives the hair the shape of a coude catheter (Bailey, Bishop & Morson, 1958). In 2010, Inui *et.al.* (85) described coudability hairs on trichoscopy and positively co-relating them with disease activity in AA.

Black dot-

It is a ‘cadaverized hair’ that has undergone necrosis due to severe inflammatory process and is retained within a hair follicle. This is visible as a ‘black dot’ by dermoscopy or

videodermoscopy. Numerous black dots are found in patients with AA and dissecting cellulitis of scalp (DCS). The mean number of black dots at 70 magnification is four in acute AA, one in chronic AA and two in DCS. In healthy people or in patients with other conditions, only incidental black dots are found (86). In a study by Kowalska-Oledzka *et.al.* (87) the black dots were seen in 53.3% patients of AA, 40% patients of severe chemotherapy-induced alopecia, and in 100% patients of dissecting cellulitis of the scalp. There were no black dots seen in patients with AGA or TE.

Flame hair-

'Flame hairs' first described by Rakowska *et.al.* as a trichoscopic sign of trichotillomania, defined as a type of hair residue that results from severe external injury to the hair shaft after pulling anagen hairs. Flame hairs are detected in 100% of the acute chemotherapy- and radiotherapy-induced alopecias, where they are the predominant finding. They are also found in trichotillomania (55%), alopecia areata (21%), traction alopecia (4%) and central centrifugal cicatricial alopecia (3%). On pathology, they corresponded to distorted hair shafts (88).

Loss of follicular units-

They indicates scarring alopecia. It is seen in all types of scarring alopecia. Others findings should be looked for differentiate between scarring alopecia. (28, 46).

Scaling-

Scales is appreciated well with dry dermoscopy. Scales can be interfollicular, perifollicular or both. Interfollicular and perifollicular scales can be seen in discoid lupus (69,89).

Telangiectasia-

These are vascular structures are seen on trichoscopy in seborrheic dermatitis, contact dermatitis, psoriasis, folliculitis decalvans, and connective tissue disorders (72,90). Arborizing vessels, branching capillaries, giant capillaries are seen in discoid lupus erythematosus (72,79). In the study by Lallas *et.al.* in 2012, decribed that telangiectatic vessels are observed in DLE lesions of longer duration (81).

Honey-comb pattern-

It is a feature of the sun-exposed or pigmented scalp and consist of a homogenous mosaic of brown, contiguous mesh around hypopigmented areas(91). The brown lines correspond to the rete ridge melanocytes, whereas the melanocytes residing in the suprapapillary epidermis results in the hypochromic areas (92). The exaggerated form of honeycomb pattern is typically seen in discoid lupus erythematosus, where it is disrupted and is observed in DLE plaques of longer duration (79,81).

Structure less white patches-

White patches on trichoscopy signify scarring alopecia in the pigmented scalp (92). They are white irregular areas devoid of follicular openings. They results due to dermal fibrosis and are evident in DLE plaque of longer duration as described by Lallas *et.al.* in 2012. (37, 38).

Short regrowing hairs-

They are short, upright, tapered regrowing terminal hairs of normal thickness. They are seen in clusters in the regrowing patches of alopecia areata, and interspersed with long terminal hairs in telogen effluvium. They are also seen in normal scalp. (32, 50). In 2009,

Adriana Rakowska *et.al.* have reported more short regrowing hair in chronic telogen effluvium than female pattern hair loss (70).

Circle hairs or pigtail hair-

They are thin coiled hairs. They are seen in alopecia areata and androgenetic alopecia. Presence of a high number of coiled hairs is highly suggestive of alopecia areata (72,93).

Exclamation mark hair-

It is a broken hair with a dark, frayed, thick tip. Its proximal portion is thin and hypopigmented. They correspond to telogen hair with a broken tip. Exclamation mark hairs are typical of alopecia areata (94). They are co-related with disease activity (95,96). In 2014, Ankad *et.al.* also reported them to be the sign of active disease. (93)

Broken hairs- They are hair shafts which are fractured at different levels from scalp emergence. They are prominent pattern in many of the nonscarring alopecias. (97).

Trichoscopy in Androgenic alopecia- Male and female AGA share similar trichoscopic features, including hair shaft thickness heterogeneity, thin hairs, yellow dots, perifollicular discoloration (the peripilar sign), an increased proportion of vellus hairs, and a large number of follicular units with only one emerging hair shaft (70,73,98,99). In 2009 Rakowska *et.al.* introduced a criteria for diagnosis of female pattern hair loss (70).

A. Major criteria

1. More than 4 yellow dots in four images at a 70-fold magnification in the frontal area.

2. Lower average hair thickness in the frontal area in comparison with the occiput (calculated from not less than 50 hairs from each area).

3. More the 10% of thin hairs (below 0.03 mm) in the frontal area.

B. Minor criteria

1. Ratio of single-hair unit percentage, frontal area to occiput >2:1

2. Ratio of number of vellus hairs, frontal area to occiput >1.5:1

3. Ratio of hair follicles with perifollicular discoloration, frontal area to occiput >3:1.

This criteria was developed after comparing the trichoscopic finding in 131 Polish female patients, 59 with female androgenetic alopecia, 33 with chronic telogen effluvium and 39 healthy volunteers. There is no validated trichoscopic criteria for diagnosing AGA in Indian population.

Trichoscopy of telogen effluvium- Trichoscopy has limited value in diagnosing telogen effluvium. The non-specific but frequent findings include the presence of empty hair follicles, a predominance of follicular units with only one hair, perifollicular discoloration (the peripilar sign), and upright regrowing hairs. There is no significant difference between the findings in the frontal area and those in the occipital area, which differentiates telogen effluvium from androgenetic alopecia (41).

Trichoscopy of anagen effluvium- due to toxicity is characterized by the presence of monilethrix-like hairs and black dots (41).

Trichoscopy in alopecia areata- The hallmark trichoscopic features of alopecia areata are regularly distributed yellow dots, micro–exclamation mark hairs, tapered hairs, black dots (formerly called cadaverous hairs), broken hairs, clustered short vellus

hairs (shorter than 10 mm) and regrowing upright or regrowing coiled hairs in the areas of hair loss . Trichoscopy of alopecia areata may differ depending on disease activity, severity, and duration (59,100). The trichoscopic findings in alopecia areata in various studies is shown in Table 5.

Table 5: Trichoscopic features in alopecia areata in various studies

Variable	**Inui *et.al.*** (74)	**Chiramel *et.al.*** (101)	**Ankad *et.al.*** (93)	**Hegde *et.al.*** (75)	**Dincy *et.al.*** (102)	**Thappa *et.al.*** (103)
Total patients	300	24	50	75	57	66
Place	Japan	North India	South India	South India	South India	South India
Yellow dots	63.70%	87.50%	50%	57.33%	42%	81.80%
Black dots	44.30%	79.20%	20%	84.00%	75%	66.60%
Broken hair	45.70%	70.80%	30%	37.33%	67%	55.40%
Short vellus hair	72.70%	50.00%	10%	68.00%	56%	40.90%
Shot regrowing hair	31.70%	Not calculated	Not calculated	18.67%	33%	12.10%

Trichoscopy in discoid lupus erythematosus- Trichoscopic features of discoid lupus erythematosus in active (early) lesions are thick arborizing vessels, large yellow dots (follicular keratotic plugs), fine interfollicular scaling, scattered brown discoloration, red dots and blue-gray dots (on dark or sun-exposed skin). While inactive lesions show loss of follicular openings, white areas, pink areas, arborizing vessels and yellow dots containing thin spider vessels (in prefibrotic lesions) (78,90,104). The trichoscopic features of DLE in various studies are summarized in Table 6. The study by Duque *et.al.* in 2010 (79), reported the blue gray dots in DLE are seen in a speckled pattern in comparison to LPP in which they have a target pattern. The study Lallas *et.al.* in 2013 reported, perifollicular whitish halo, follicular keratotic plugs and telangiectasias were the most common dermoscopic criteria with a frequency of 69.1%, 67.3% and 52.7%, respectively (81).

Thakur *et.al.* from Northeast India in 2015 studied DLE trichoscopic features in 10 patients and found yellow dots in 70%, black dots in 20%, loss of follicular units in 100%, hyperkeratotic plugs in 90%, telangiectasias in 80%, brown discoloration in 70%, structureless white patches in 100%, blue gray dots in 20%, perifollicular erythema in 100%, perifollicular scaling in 80% and epidermal atrophy in 100% of the patients (42). Hyperkeratotic plugs were the most common finding in the study by Hashem *et.al.* 2015 (77).

Table 6: Trichoscopic features of DLE in various studies

Variable	**Lallas *et.al.*** (81)	**Duque *et.al.*** (79)	**Thakur *et.al.*** (42)	**Hashem *et.al.*** (92)
Total Patients	55	5	10	5
Hyperkeratotic Plugs	67.30%	100%	90%	100%
Red Dots or Globules	36.40%	NA	NA	40%
Telangiectasias	52.70%	100%	80%	80%
Scaling	49.10%	0%	80%	40%
Exaggerated HCP	43.60%	40%	70%	NA
Perifollicular White Halo	69.10%	NA	NA	NA
Structureless White Area	36.40%	100%	100%	40%
Loss Of Follicular Units	NA	80%	100%	Not specified
Blue Gray Dots And Globules	NA	40%	20%	Not specified
Blue/Gray/Brown Speckled Pigmentation	NA	NA	NA	20%
Yellow Dots	NA	NA	70%	20%

8. TRICHOSCAN OF NON-SCARRING DIFFUSE ALOPECIA

Trichoscan is carried out by an in-built software in the videodermoscopes which allows to carry out measurements of structures visualized in magnified photographs and provides results in real scale (70,105).

In 2009, Rakowska *et.al.*, reported in Polish population, the *average hair thickness* in frontal area and occiput as 0.061 ± 0.008 mm and 0.058 ± 0.007 mm respectively in healthy controls. It was 0.047 ± 0.007 mm and 0.052 ± 0.008 mm respectively in androgenic alopecia. And it was 0.056 ± 0.007 mm and 0.053 ± 0.009 mm respectively in TE.

Mean percentage of vellus hair (< 0.03 mm) was 20.9 ± 12% in androgenic alopecia, 10.4 ± 3.9% in telogen effluvium and 6.15 ± 4.6% in healthy individuals.

Mean percentage of single-hair pilosebaceous units in healthy individuals is 27.3 ± 13% and 22.6 ± 12.6% in frontal and occipital scalp respectively. In AGA, it was 65.2 ± 19.9% in the frontal scalp and 36.8 ± 18.6% in the occipital scalp and TE, it was 39.0 ± 13.4% and 31 ± 23% in the frontal and occipital scalp respectively.

Mean percentage of perifollicular discoloration in FAGA was 32.4 ± 4.7% in the frontal area and 6.6 ± 2% in the occipital area (70).

Terminal verses vellus ratio is 8:1 in healthy individuals. The ratio of less or equal to 4:1 is considered as FAGA. The *anagen verses telogen ratio* is lower than 6.6 in telogen effluvium. Normally there are 86% of hair are in anagen stage, 13% in the telogen stage and rest in the catagen stage. This ratio is normal in FAGA (70,106).

9. MERITS AND DEMERITS OF VIDEODERMOSCOPY AND TRICHOSCAN

1. Trichoscopy is very fast and provide highly instructive clues towards the diagnosis of hair and scalp disorders.
2. Videodermoscopy is simple and highly descriptive for hair and scalp analysis. It can be served as a step prior to performing a biopsy and can help to find the right place to take the sample, and thus avoid unnecessary biopsies.
3. Trichoscan is simple, painless and speedy photographic processing with the reproducibility of results. But there have been disputes regarding the accuracy and the TrichoScan software is error prone and not precise (107).
4. Saraogi and Dhurat in 2010 (108), studied the utility of trichoscan in quantification of diffuse hair loss and concluded that this is an overstatement of this procedure. This also concludes that anagen/telogen hair detection by TrichoScan is not optimal and there is overestimation of total hair density and telogen hair percentage. And also the vellus hair percentage does not correlate with clinical severity of alopecia.

10. DISEASE ACTIVITY IN SLE

Measurement of disease activity in SLE is significant in evaluating differences among SLE patient groups, outcomes, responses to new proposed drugs, and for assessing disease longitudinally for observational studies and clinical trials (109).

Two types of activity measures in SLE have been developed:

1. Global score systems (e.g. Systemic Lupus Activity Measure [SLAM], the European Consensus Lupus Activity Measurements, and Systemic Lupus Erythematosus Disease Activity Index [SLEDAI]). They provide an overall measure of activity.
2. Individual organ/system assessment scales- They assess disease activity in single organs such as the British Isles Lupus Assessment Group Index [BILAG]. The Systemic Lupus International Collaborating Clinics (SLICC) or American College of Rheumatology Damage Index score is a measure for chronic damage. It has been included due to its prognostic value in clinical and research basis (10).

The original version of SLEDAI was introduced in 1985 (11). In 2002, it was modified to reflect persistent active disease in those descriptors that had previously considered new or recurrent occurrences (SLEDAI-2K) (12). There are twenty four items for the nine organs/systems. Score range from 0–105 points (10).

Activity categories have been defined on the basis of SLEDAI scores as follows (110):

1. No activity (SLEDAI = 0)
2. Mild activity (SLEDAI = 1-5)
3. Moderate activity (SLEDAI = 6-10)
4. High activity (SLEDAI = 11-19)
5. Very high activity (SLEDAI 20).

Flare of SLE is defined as an increase in SLEDAI > 3 and if SLEDAI score > 5 indicates probability of initiating or changing therapy in more than 50% of instances (111).

The skin lesions are correlated with diasease activity as shown in the study by RD ZecÏevicÂ *et.al.* in 2001. This study showed that LE-nonspecific lesions have significantly more active disease than those with LE-specific lesions (SLEDAI-7.25±4.27 verses 7.34±3.60 respectively). It was also shown in this study that number of skin lesions also increase with SLEDAI activity. Patients with one, two and three or more skin lesions were found to have mean SLEDAI of 7.15, 12.26 and 18.50 respectively.

In this proposed study, we intent to describe type of alopecia with trichoscopic features, in patients with SLE and there correlation to the disease activity. To the best of our knowledge, even after extensive literature search we could not find any studies describing the clinical patterns and trichoscopic patterns of alopecia in systemic lupus erythematosus. This study will throw light on this topic and also will show its co-relation with severity of disease.

MATERIALS AND METHODS

Study design

The study was a hospital based cross sectional observational study, of patterns of hair loss in patients with systemic lupus erythematosus (SLE). Patients with SLE attending the outpatient departments in dermatology / rheumatology or admitted under these departments were included in the study.

Setting

The study was conducted in the Department of Dermatology, Venereology and Leprosy Unit -2 at Christian Medical College, Vellore, a tertiary care hospital in Vellore, Tamil Nadu.

Study duration

The study was conducted between September 2016 and August 2017 (12 months).

IRB approval - The study was approved by the Institutional Review Board. (IRB approval no -10266)

Inclusion criteria:

1. Patients with a diagnosis of Systemic Lupus Erythematosus based on SLICC criteria.
2. Patients aged > 18 years and willing to participate in the study.

Exclusion criteria:

1. Patients who are not willing to participate in the study

2. Age less than 18 years.

Sample size calculation-

Formula

$$n = \frac{Z^2_{1-\alpha/2}\, p(1-p)}{d^2}$$

Where,

p : Expected proportion

d : Absolute precision

1- α/2 : Desired Confidence level

Expected Proportion	0.6
Precision (%)	10
Desired confidence level (1- alpha) %	95
Required sample size	***92***

The prevalence data used for the calculation of sample size was from the study by Yun SJ *et.al.* in 2007, in which hair loss was seen in 20-60% patients with systemic lupus erythematosus (28).

Methodology

Patients with established diagnosis of SLE were enrolled in the study (new patients as well as the follow up patients who were already on treatment) after obtaining their informed consent. The demographic details were recorded including the age, sex, address

and marital status. History of the duration of disease (from the age of onset till inclusion in the study), duration of the hair loss, pattern of hair loss (patchy / diffuse or both), approximate amount and course of hair loss and any triggers or recent exacerbations were recorded. The association of hair loss with symptoms of SLE were asked for, and any improvement with SLE medications were recorded. Other causes of hair loss were ruled out by asking past or present history of thyroid abnormalities or other comorbidities, association with preceding illness, past surgeries or stress in last 6 months, diet, food habits, and frequency of hair wash in a week, and use of cosmetic or chemical procedure done for hair. History of skin, scalp, and mucosal involvement and any history suggestive of any organ involvement were asked for. A detailed drug history of use of topical steroids, topical calcineurin inhibitor, sunscreen, antimalarial, dose and type of oral steroid and adjuvant drugs were noted. The patient's drug history was corroborated by the hospital's electronic pharmacy records. The vital signs were recorded and clinical examination was done including examination of skin, mucosa, nails and joints.

The patients were examined for the type of hair loss and they were classified clinically as having-

- Diffuse non-scarring alopecia
- Patchy non-scarring alopecia
- Scarring alopecia
- A combination of the above

Clinical diagnosis of alopecia:

1. A clinical diagnosis of *androgenetic alopecia* was made on the basis of rarefaction in mid- section with the preservation of frontal hair line in females and receding frontal hair line or bi-temporal recession in males. The terminology androgenetic alopecia was used as the study included both the genders.
2. Clinical diagnosis of *alopecia areata* was based on the basis of patchy non-scarring hair loss or total alopecia of scalp without the features of scarring.
3. Clinical diagnosis of *DLE* was made on the basis of clinical scarring alopecia with smooth hypopigmented atrophic plaque with or without adherent scaling. The additional features looked for were peripheral hyperpigmentation and presence of DLE lesions on the areas of the body.
4. Clinical diagnosis of *anagen effluvium* was considered if there was a history of sudden onset extensive hair loss from the whole scalp after initiation of medications.
5. The clinical diagnosis of *telogen effluvium* was made when patient presented with history of hair loss with the absence of any other type of alopecia mentioned above. The telogen effluvium was further classified on the basis of history, as acute telogen effluvium if hair loss is present for more than 3 months, and chronic telogen effluvium if hair loss is present for less than 3 months.

Trichoscopic evaluation:

Trichoscopy was done in all the patients with alopecia and trichoscopic photographs were obtained using a FotoFinder Systems GmbH (software version from 2.1). FotoFinder (FotoFinder Systems GmbH, Bad Birnbach, Germany) videodermatoscope is an immersion dermoscopy nonpolarized system equipped with a videocamera with lenses providing magnifications ranging from 20× to 120×. The images obtained are visualized on a monitor and stored on a computer. In patients with diffuse non-scarring alopecia, trichoscopy was done at four sites- frontal, both temporal areas and occipital area. Trichoscopy was also done on the specific lesional site of alopecia areata or DLE. Photographs were obtained at 20X and 70X magnification. Whenever, trichoscopic photographs at the 70X magnification were not clear, other appropriate magnifications < 70X were used.

Trichoscan was done in case of diffuse non-scarring alopecia i.e. androgenetic alopecia and telogen effluvium. Trichoscan was done from the most representative site of alopecia on the frontal and occipital region. Hair clipping could not be done in our patient group as the patients were not willing and were more concerned about their primary disease i.e. SLE. Trichoscan was done with an in built software in the FotoFinder Systems GmbH (software version from 2.1). The following measurements were recorded:

1. Hair count
2. Hair density (in cm^2)
3. Vellus hair percentage

4. Vellus hair density (in cm^2)
5. Single follicular units (percentage)
6. Average hair thickness (in mm)
7. Ratio of telogen and vellus hair

As hair clipping was not done, ratio of anagen and telogen hair was not recorded.

Trichoscopic criteria for diagnosis of alopecia:

1. *Female androgenic alopecia:* The diffuse non-scarring alopecia was classified as AGA if hair diameter diversity was more than 20% at 20X magnification.
2. *Telogen effluvium:* The diffuse non-scarring alopecia was classified as telogen effluvium if there were no features to suggest AGA.

   The other trichoscopic features looked for in diffuse non-scarring alopecia were:

a) Vellus hair more than 10% / Less than 10% vellus hair at 20X magnification
b) Presence or absence of predominant single hair follicular units at 20X magnification
c) Absence or less than 20% hair diameter variability.
d) Yellow dots (scanty – if less than 3; numerous – if 3 or more at 20X)
e) Perifollicular discoloration (if 3 or more follicular units showed peripilar sign at 20X)
f) Ratio of terminal verses vellus hair on trichoscan.

3. *Alopecia areata*: It was mainly a clinical diagnosis. The trichoscopic features looked for were as follows.

a) Yellow dots (scanty – if less than 3; numerous – if 3 or more at 20X)
b) Multiple black dots at 20X

c) Exclamation mark hair

d) Broken hair

e) Regrowing circles or pigtail hairs

f) Flame hair

4. *Discoid lupus erythematosus*: For trichoscopy the most representative site of DLE was chosen. The trichoscopic features looked for were as follows.

a) Perifollicular white halos

b) Follicular hyperkeratotic plugging

c) Telangiectasia

d) Structureless white areas

e) Loss of follicular units

f) Red dots or globules

g) Blue gray dots or globules

h) Blue gray speckled pigmentation

i) Large yellow dots

j) Interfollicular scaling

k) Exaggerated honey comb pigment network

5. *Anagen effluvium:* The following points were considered for diagnosis of anagen effluvium, if the patients had sudden onset hair loss after initiation of medication.

a) Yellow dots

b) Black dots

c) Flame hairs

d) Acute constrictions

e) Color changes along the hair shaft

f) Tapering hairs

g) Caudability hairs

Trichoscopic observations were recorded by the primary investigator and then were reviewed and confirmed by the guide.

The final diagnosis of diffuse non-scarring pattern of alopecia was made by clinical examination and trichoscopic findings at each site – frontal and occipital. Although trichoscopic images of temporal region were taken, it was not considered for making the final diagnosis as studies have shown that percentage of single follicular units are more in the temporal scalp (112). In patients with discordant trichoscopic findings in the frontal and occipital scalp in case of diffuse non-scarring alopecia, a diagnosis of combination i.e. AGA and TE; was made.

The percentage of scalp involvement in AA and DLE was calculated, using the percentage of scalp involvement as described in the SALT score (Annexure 5). In patients showing features of more than one type of alopecia (diffuse or patchy non-scarring and scarring alopecia), diagnosis of each type was recorded.

The disease activity was calculated using Systemic lupus erythematosus disease activity index (Annexure 6) by the primary investigator and cross checked with electronic records later (SLEDAI calculated and recorded in the discharge summary or out patient record by the rheumatologist in that admission or visit).

Investigation

Investigations to rule out other causes of alopecia were done like Iron, Ferritin, Vitamin B12, Vitamin D and thyroid stimulating hormone (TSH). Latest haemoglobin, total count, differential count and platelet counts were recorded form the electronic records of the patient.

Statistical methods

Descriptive statistics were used such as number and percentage for categorical variable and mean, standard deviation or median and interquartile range (IQR) were used for all the continuous variables. However, histogram was done for continuous variables to study the distribution. When the histogram suggested normal distribution across the groups, independent T- test and ANNOVA test were done. And if the distribution was not normal Mann- Whitney U test and Kruskal-Wallis test were used. Chi-square test was used to assess the association between two categorical variables. P value at 5% level significance was considered as statistical significant. The data was entered using Epidata version 3.1, and analysis was done using the SPSS software (Version 21).

RESULTS

One hundred patients with suspected SLE were included during the period of study from September 2016 to August 2017. Four patients were excluded, as three of the patients did not fulfill the SLICC criteria and the fourth patient was below 18 years. There were 96 patients who consented to participate in the study.

1. DEMOGRAPHIC PROFILE OF PATIENTS:

1.1 Age and sex distribution of patients.

There were 83 (86.5%) females and 13 (13.5%) males (M: F ratio 1: 6.6) as shown in Figure 2. Majority of patients were in the 20-39 age group. (Table 7)

Table 7: Age and sex distribution in patients with SLE

Age range	Female	Female (n)	Male	Male (n)
18- 20 years	3.61%	3	23.07%	3
20-39 years	73.49%	61	46.15%	6
40-59 years	22.89%	19	30.76%	4

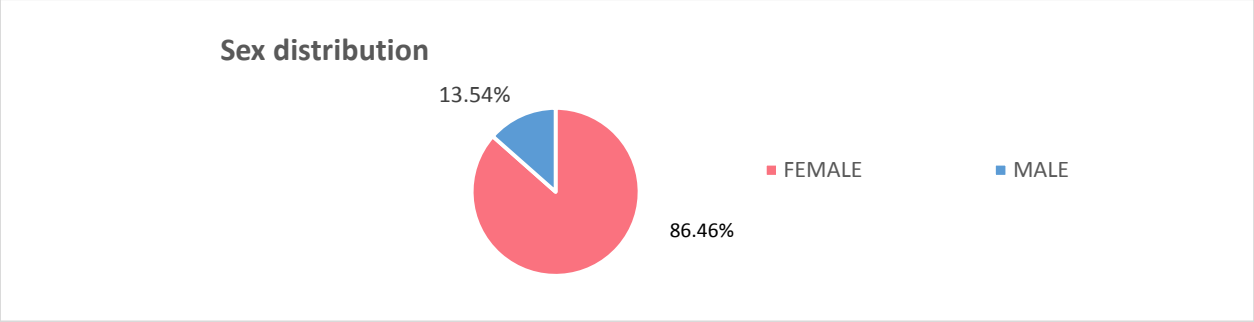


Figure 2: Gender distribution of patients with SLE

1.2 Age of onset of SLE:

The mean age of onset of SLE in females was 27.99± 9.66 years and in males it was 26.54 ±9.483 years. There was no significant variation in the age of onset between the two groups (P = 0.6151).

1.3 Geographical distribution of patients:

The geographical distribution of the study patients are shown in Figure 3. The majority of the patients were from the Tamil Nadu (40.6%) followed by West Bengal (24%), Jharkhand (11.5%), Andhra Pradesh (6.3%), Bihar (5.2%), Kerala (4.2%) and Northeast India (3.1%). Five patients (5.2%) were not Indians (four were from Bangladesh and one from Nepal).

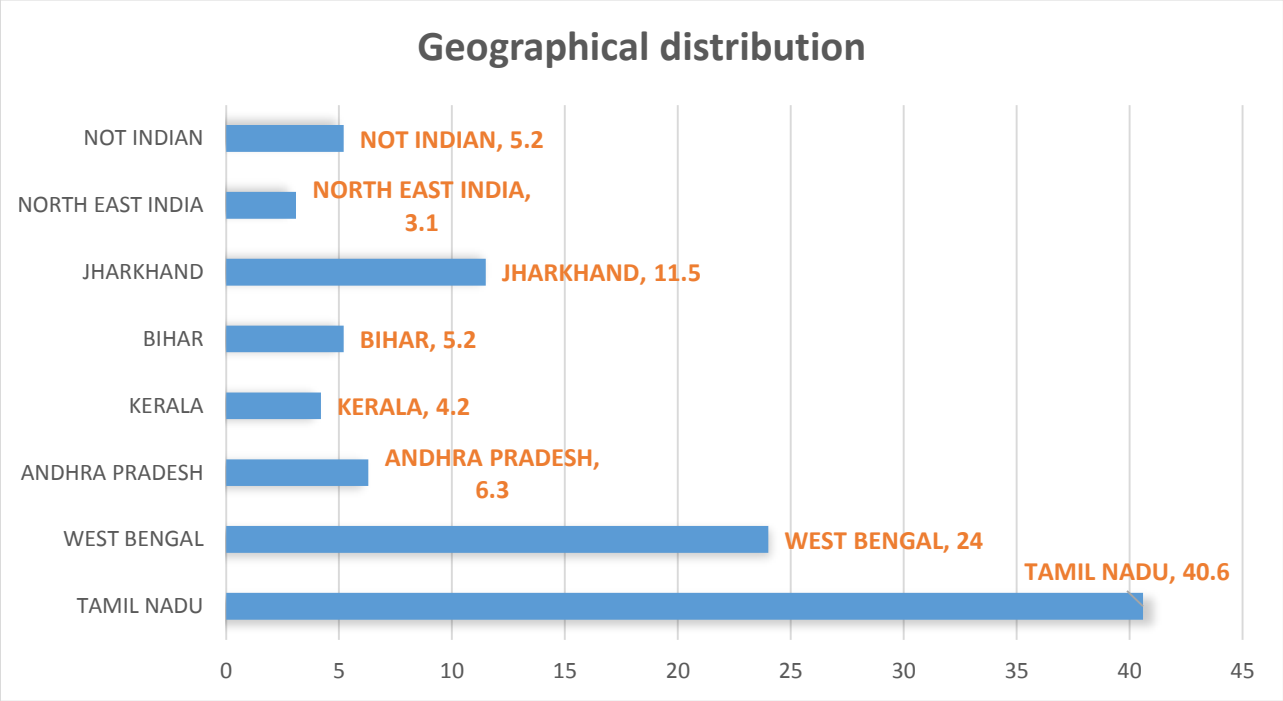


Figure 3: Geographical distribution of the patient of SLE in the study.

2. CLINICAL PROFILE OF THE PATIENTS

2.1 Duration of the SLE disease and hair loss

The mean duration of disease at presentation was 4.33 ± 4.37 years and it ranged from 1 week to 18 years. The duration of hair loss in SLE patients ranged from 1 week at presentation to 25 years at the time of inclusion in the study. The mean duration of alopecia at presentation was mean of 3.39 ±4.52 years. There were 14 (14.6%) patients who had history of hair loss even before the diagnosis of SLE.

2.2 Amount of hair loss and pattern of hair loss as per the history:

The history of amount of hair loss per day given by the patients is shown in Figure 4. Majority (69.8%) of patients gave history of hair loss of more than 100 hairs per day.

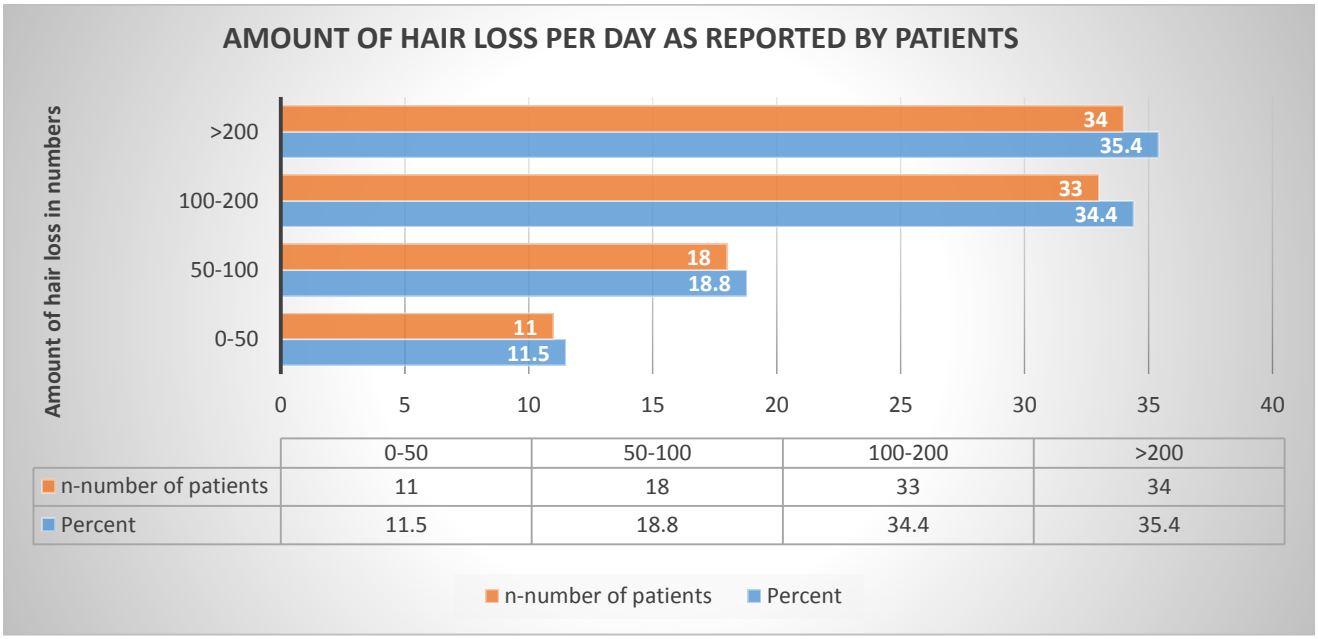
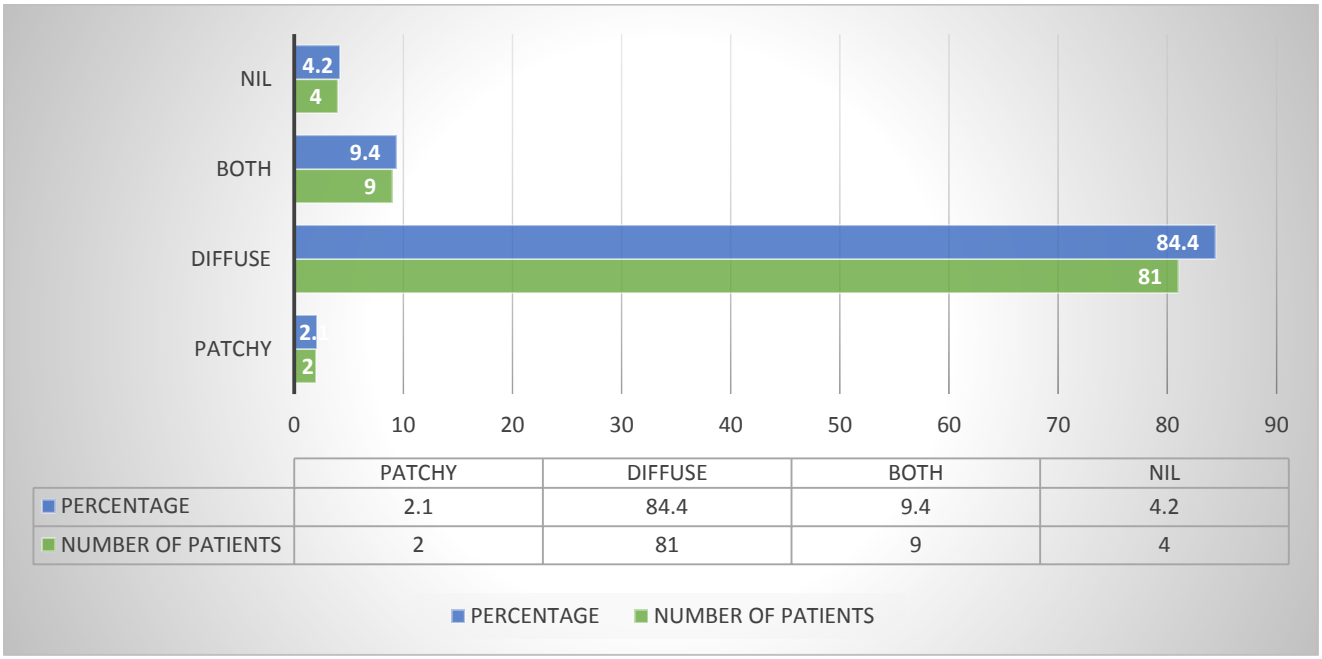


Figure 4: Amount of hair loss per day as reported by patients.

Among the patient with history of episodic hair loss (22 patients i.e. 22.9%), 16 patients (16.7%) noticed some trigger preceding the periods of increased hair loss in the form of fever, erythematous skin lesions, increased joint pains etc. Among all patients, 76 patients (79.2%) noticed the increase in hair loss at the time of disease flare.

The various patterns of hair loss as described by the patients is shown in Figure 5. Diffuse pattern of hair loss was the most common pattern of hair loss experienced by our patients.



	PATCHY	DIFFUSE	BOTH	NIL
PERCENTAGE	2.1	84.4	9.4	4.2
NUMBER OF PATIENTS	2	81	9	4

Figure 5: Patterns of alopecia as described by the patients

2.3 History of cutaneous involvement:

Among 96 patients, 83 patients (86.5%) had history of skin involvement. Among them 12 patients (12.5%) gave history suggestive of localised cutaneous involvement (history of

involvement of skin above the level of chin) and 71 patients (74%) gave history suggestive of generalized cutaneous involvement (history of involvement of skin below the level of chin). Thirty six patients (37.5%) had history of only skin lesions without scalp involvement, one patient had history of only scalp involvement without skin lesions and 45 patients (46.9%) gave history of both skin and scalp involvement. The history of photosensitivity, oral ulcer and nasal ulcer, was present in 59 (61.5%), 67 (69.8%) and 29 (30.2%) patients respectively.

2.4 Systemic manifestations:

The most common systemic manifestation was joint pain (76%) followed by renal involvement (55.2%), chest pain (25%), breathing difficulty (24%) and abdominal pain (14.6%).

2.5 Comorbidities in the patients

The most common comorbidity among SLE patients was thyroid disease (25%) followed by hypertension (9.4%) and tuberculosis (9.4%), diabetes mellitus (5.2%).

2.6 Personal and family history with regard to hair loss

Personal history:

There were 35 female patients with menstrual irregularities (42.16%; 35/83). Five patients (5.2%) were on weight reducing diet either due to obesity or secondary cushingoid habitus due to corticosteroid therapy. Eighty six (89.6%) patients had mixed

diet, 7 (7.3%) were vegetarians and 3 (3.1%) were non-vegetarians. Cosmetic hair procedures were practiced only by a very few patients. Seven percent (7.3%) of the patient had chemically colored hair at the time of presentation and six patients (6.3%) had done chemical straightening.

Family history:

There were 3 patients (3.1%) who had positive family history of SLE. Twelve patients (12.5%) had history of hair loss in family due to causes other than SLE.

2.7 Ongoing Therapy for SLE among patients

Topical medicaments:

There were sixty seven patients (69.8%) who were using sunscreen at the time of presentation. Forty five patients (46.9%) and 29 patients (30.2%) had used topical steroids and topical calcineurin inhibitors one or more times during the period of disease for muco-cutaneous involvement.

Oral steroids:

There were 88 patients (91.7%) who were using oral steroids. Majority of the patients (54.2%) were on deflazacort followed by 34.5 % of the patients on prednisolone and 2.1% of the patients on dexamethasone.

Adjuvant immunosuppressant drugs and antimalarials :

There were 78.1% of patients who were on adjuvant drugs at the time of inclusion in the study as shown in Figure 6. Ninety one patients (94.8%) were on hydroxychloroquine, one patient was on chloroquine and 4 patients (4.2%) were not taking antimalarials at the time of inclusion in the study.

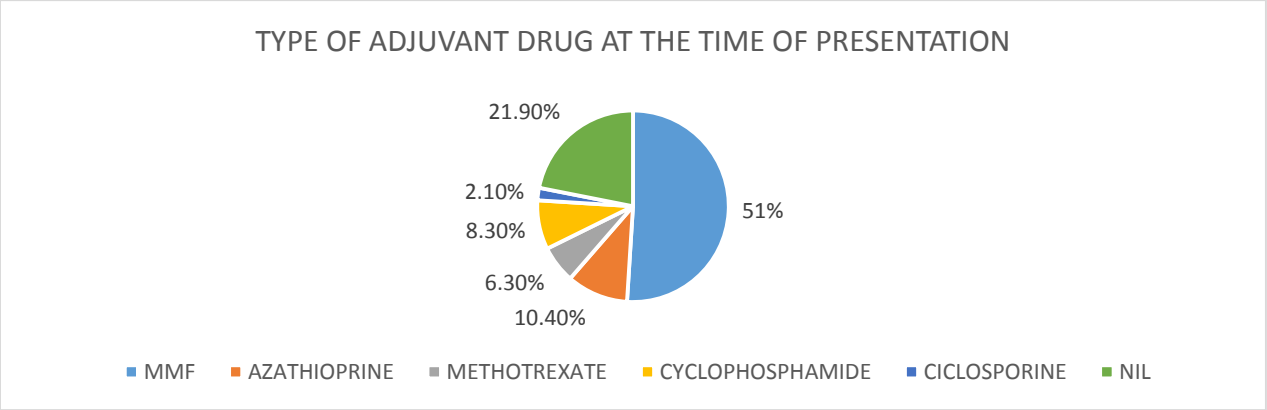


Figure 6: Type of adjuvant drugs at the time of presentation

3. MUCOCUTANEOUS LESIONS IN PATIENTS

3.1 Specific and non-specific cutaneous lesions of LE.

Forty patients (41.7%) presented with acute cutaneous lupus erythematosus. Among them, 18 patients (18.8%) had localised malar rash and 22 patients (22.9%) had generalised rash. Four patients (4.2%) presented with SCLE rash (papulosquamous skin lesions). Twenty one patients (21.9%) presented with chronic cutaneous lupus erythematosus. Among them 19 patients (19.8%) had classic discoid lupus erythematosus, one patient had lupus panniculitis. The types of cutaneous LE in study group is shown in Figure 7.

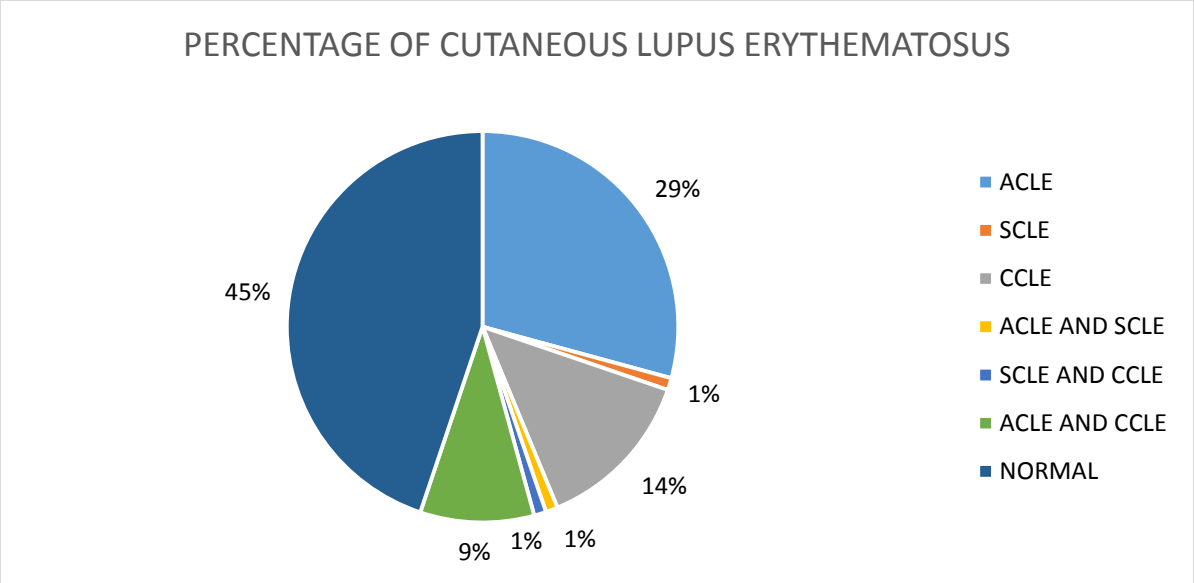


Figure 7: Types of cutaneous lupus erythematosus in patients.

3.2 *Mucosal examination:*

Mucosal examination of the patients is shown in the Table 8. There were 18 patients with oral ulcers and 4 patients with nasal ulcers

Table 8: Mucosal findings of patients

Mucosae	Number of patients	Percentage
Oral ulcer	18	18.8%
Nasal ulcer	4	4.2%
Conjunctival ulcer	0	0%

4. ALOPECIA

Age of onset of hair loss in diffuse non-scarring alopecia

The age of onset among diffuse non-scarring alopecia (n=78) was between 18-30 years in 65.38% patients, between 31-40 years in 21.79% patients, between 41-50 years in 11.53% patients and between 51-60 years in 5.26% patients.

Clinical Patterns of alopecia in SLE patients

Majority of the study population had diffuse non-scaring alopecia (81.25%). Among the diffuse non-scarring alopecia, 14 patients (14.58%) had clinically evident AGA and 14 patients (14.58%) had lupus hair. Table 9 shows the frequency of various types of alopecia among the study population.

Table 9: Frequency of different clinical patterns of alopecia

Specific types of alopecia	**Number of patients**
Diffuse non-scarring alopecia	78 (81.25%)
DLE	26 (27.08%)
Lupus hair	14 (14.6%)
Patchy non-scarring alopecia (alopecia areata)	6 (6.25%)
Anagen effluvium	0

The combination of these patterns were observed in 14 patients (14.58 percentage) as shown in Table 10.

Table 10: Combinations of clinical pattern of alopecia (non-scarring and scarring)

Combinations of clinical patterns of alopecia (non-scarring and scarring)	**Number of patients**
Diffuse non-scarring alopecia	64 (66.66%)
DLE	16 (16.66%)
Diffuse non-scarring alopecia and DLE	10 (10.41%)
Diffuse non-scarring alopecia and alopecia areata	4 (4.16%)
Alopecia areata	2 (2.08%)
Total	**96**

4.1. TRICHOSCOPY

A. Trichoscopic examination in diffuse non-scarring alopecia

Trichoscopy was done from the frontal and occipital scalp in all the patients with diffuse non-scarring alopecia. There were 78 patients (81.25%) with diffuse non scarring alopecia. In 13 cases, either the frontal or occipital scalp could not be assessed as the patient had alopecia areata or DLE on frontal or occipital scalp region. Based on the trichoscopy, the patterns of diffuse non-scarring alopecia were categorised into androgenetic alopecia, telogen effluvium, combination of the two or anagen effluvium. The distribution of various trichoscopic patterns of diffuse non-scarring alopecia in our study are shown in Table 11.

Table 11: Diffuse non-scarring alopecia- trichoscopic patterns

PATTERN	FREQUENCY	PERCENTAGE
AGA	19	19.79%; (19/96)
TE	46	47.91%; (46/96)
AGA+TE	13	13.50%; (13/96)
Total Diffuse Non-Scarring Alopecia	78	81.25%; (78/96)

After trichoscopy, there were 19 patients diagnosed with AGA, 46 patients with TE and 13 patients with combination of AGA and TE.

1. Comparison of age of onset among patients with diffuse non-scarring alopecia

Age comparison of AGA with TE in our study is shown in Table 12. The majority (2/3rd) of patients with AGA and TE had an early onset of alopecia between 18-30 years.

Table 12: Age comparison of AGA with TE in our study

Age of onset	**AGA (n; %)**	**TE (n; %)**	**AGA+TE (n;%)**
18-30 years	**13; 68.42%**	**32; 69.56%**	**6; 46.15%**
31-40 years	4; 21.05%	8; 17.39%	5; 38.46%
41-50 years	1; 5.26%	6; 13.04%	2; 15.38%
51-60 years	1; 5.26%	0; 0%	0; 0%
TOTAL	**19**	**46**	**13**

2. Laboratory investigation of patients with diffuse non-scarring alopecia

Haemoglobin was low among all the patients. Ferritin levels were lowest among the AGA +TE group of patients, borderline in TE group and normal in the AGA group of patients. Vitamin B12 and TSH levels were normal in all the patients. The mean investigations in diffuse non-scarring alopecia is shown in Table 13.

Table 13: Laboratory investigation of patients

Investigation	AGA	TE	AGA+TE	Normal range
Haemoglobin (gm/dl)	9.8 ±2.01	10.9 ± 2.06	10.3 ± 2.96	11-16.9g/dl (113)
Iron (ug%)	51.41 ± 33.62	48.8 ± 30.20	67.55 ± 80.49	M 60-160; F 40-145
Ferritin (ng/ml)	364.47 ± 382	298.61 ± 600	173 ± 306	M & F>50y: 20-320; F<50y:10-290
Vitamin B12 (pgm/ml)	498 ± 436	739 ± 716	781 ± 695	200-950
Vitamin D (ng/ml)	21.9 ± 12.4	28.4 ± 14.9	13.4 ± 13.4	>30
TSH (µiu/ml)	2.5 ± 1.98	2.8 ± 2.15	1.79 ± 1.34	0.3-4.5

3. Trichoscopic examination of Androgenetic alopecia

There were total 32 patients with androgenetic alopecia (AGA-19 and AGA+TE-13). Among these patients there were 29 patients who had hair diameter diversity (HDD) more than 20 percent in frontal scalp, and similarly 16 patients had HDD in occipital scalp.

Table 14: Trichoscopic findings in AGA

Trichoscopic features	Frontal (n=29) (%)	Occipital(n=16)(%)
Hair diameter diversity	29, (100%)	16, (100%)
Single follicular units	18, (62.10%)	8, (50%)
Short regrowing hair	13, (44.80%)	5, (31.30%)
Peripilar sign	6, (20.70%)	4, (25%)
Vellus hair	5, (17%)	2, (12.5%)
Scanty yellow dots(<3 dots)	5, (17%)	3, (18.75%)
Numerous yellow dots(>3 dots)	3, (10.30%)	1, (6.25%)

As shown in Table 14, the vellus hair more than 10 percent at 20x magnification, single follicular units and short regrowing hair were seen in majority of the patients, and peripilar sign and yellow dots were seen in less than 20% of the patients.

4. Trichoscopic examination of Telogen effluvium

Among patients with diffuse non-scarring alopecia (n=78), there were 59 patients (TE-46, AGA+TE- 13) with telogen effluvium. Based on the history 5 patients (8.47%) had acute telogen effluvium and 54 patients (91.53%) had chronic telogen effluvium. Among 5 patients with acute telogen effluvium, one patient had generalised acute cutaneous lupus erythematosus with cutaneous small vessel vasculitis and SLEDAI score of 24.

Another one patient with acute telogen effluvium had cutaneous small vessel vasculits with SLEDAI score of 33. Trichoscopic findings of patients diagnosed as telogen effluvium is shown in the Table 15.

Table 15: Trichoscopic findings in telogen effluvium

Trichoscopic findings	Frontal (n=46)	Occipital (n=54)
Short regrowing hair	21, 45.70%	24, 44.40%
Peripilar sign	10, 21.70%	11, 20.40%
Scanty yellow dots	3, 6.50%	4, 7.40%
Numerous yellow dots	2, 4.30%	0, 0%

As shown in Table 15, there were 46 patients who did not have HDD more than 20% or alopecia areata or DLE in frontal scalp. Similarly 54 patients did not have the above features in occipital scalp. A diagnosis of telogen effluvium was made in these patients. The majority of the patients had short regrowing hair and only few patients had yellow dots.

5. Comparison of specific trichoscopic findings of AGA and TE

a) <u>Short regrowing hair</u>

Short regrowing hair were more in the telogen effluvium (52.56%) than androgenetic alopecia (40.6%) (p= 0.275). However, this was not statistically significant. (Figure 8)

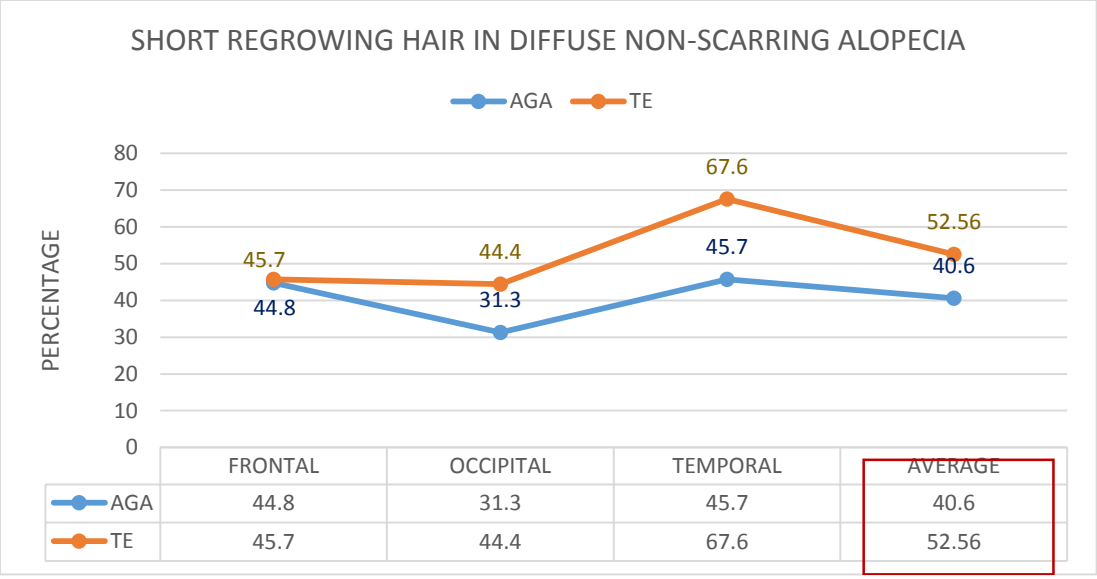


Figure 8: Short regrowing hair in diffuse non-scarring alopecia

b) *Peripilar sign*

The prevalence of peripilar sign on trichoscopy was found similar in androgenetic alopecia (18.10%) and telogen effluvium (17.63%) (p=0.962).

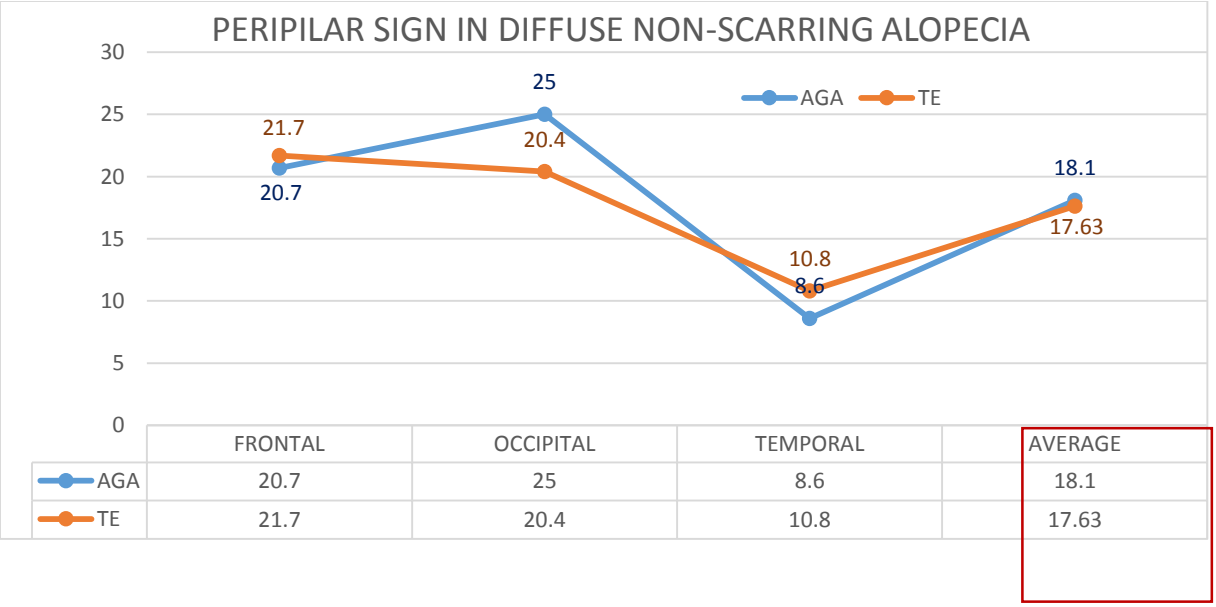
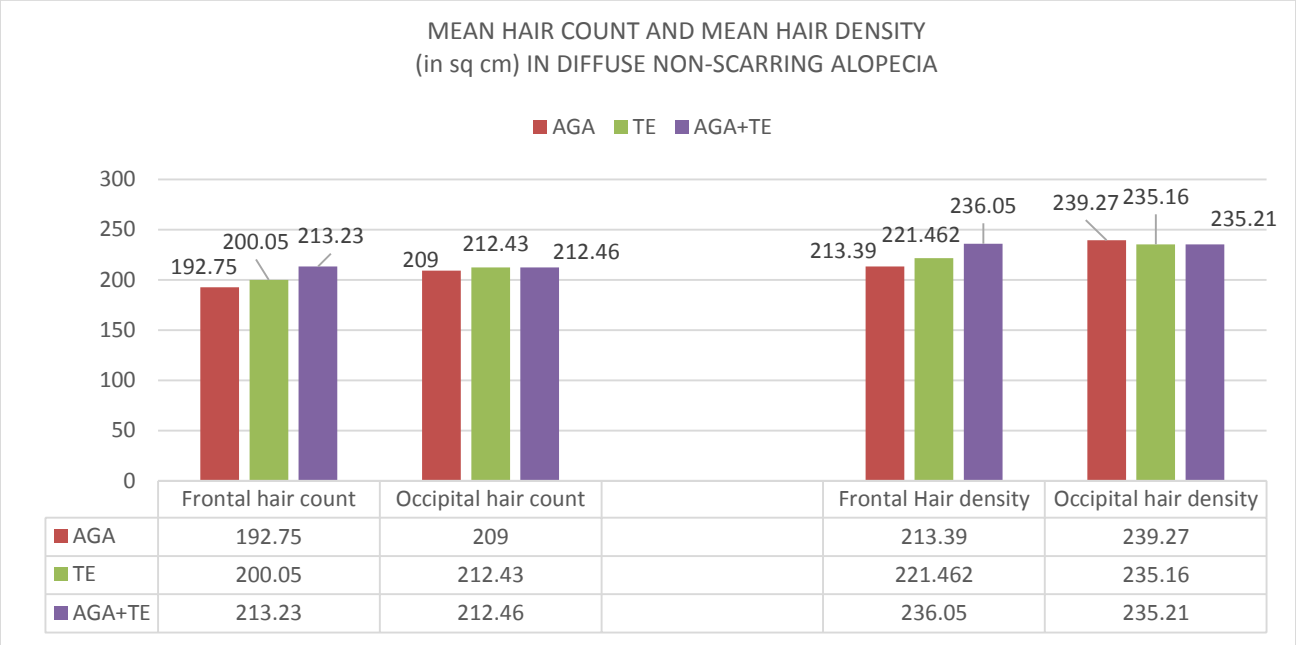


Figure 9: Peripilar sign in diffuse non-scarring alopecia

6. Trichoscan in diffuse non-scarring alopecia in our study population-

Trichoscan was done for patients with diffuse non-scarring alopecia i.e. female or male pattern androgenetic alopecia and telogen effluvium from the most representative site to support our diagnosis.

1. *Hair count and hair density in non-scarring alopecia:*

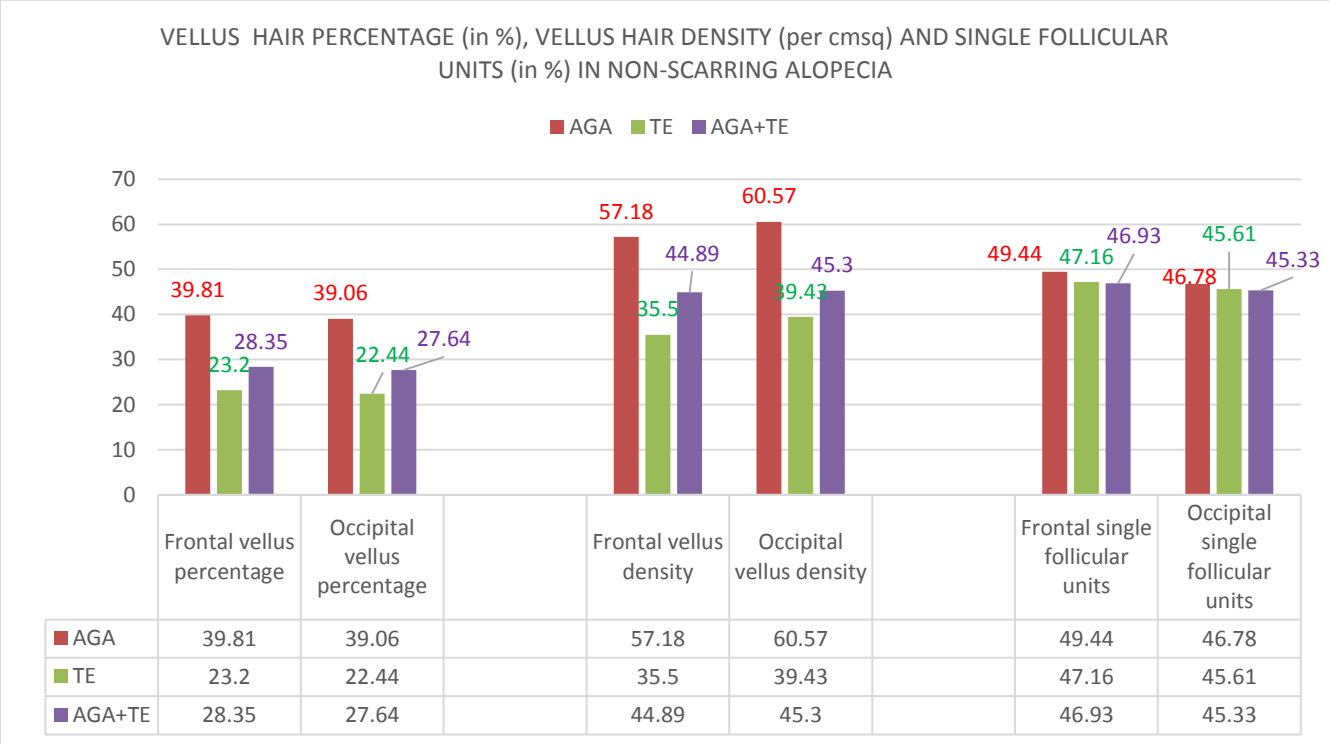


	Frontal hair count	Occipital hair count		Frontal Hair density	Occipital hair density
AGA	192.75	209		213.39	239.27
TE	200.05	212.43		221.462	235.16
AGA+TE	213.23	212.46		236.05	235.21

Figure 10: Mean hair count and mean hair density in diffuse non-scarring alopecia

As shown in the Figure 10 the mean hair count was found to be lower in AGA than TE in both frontal and occipital scalp. The mean hair density was also found to be lower in AGA than TE in frontal scalp.

2. *Vellus hair percentage, vellus hair density and single follicular units in non-scarring alopecia.*



	Frontal vellus percentage	Occipital vellus percentage		Frontal vellus density	Occipital vellus density		Frontal single follicular units	Occipital single follicular units
AGA	39.81	39.06		57.18	60.57		49.44	46.78
TE	23.2	22.44		35.5	39.43		47.16	45.61
AGA+TE	28.35	27.64		44.89	45.3		46.93	45.33

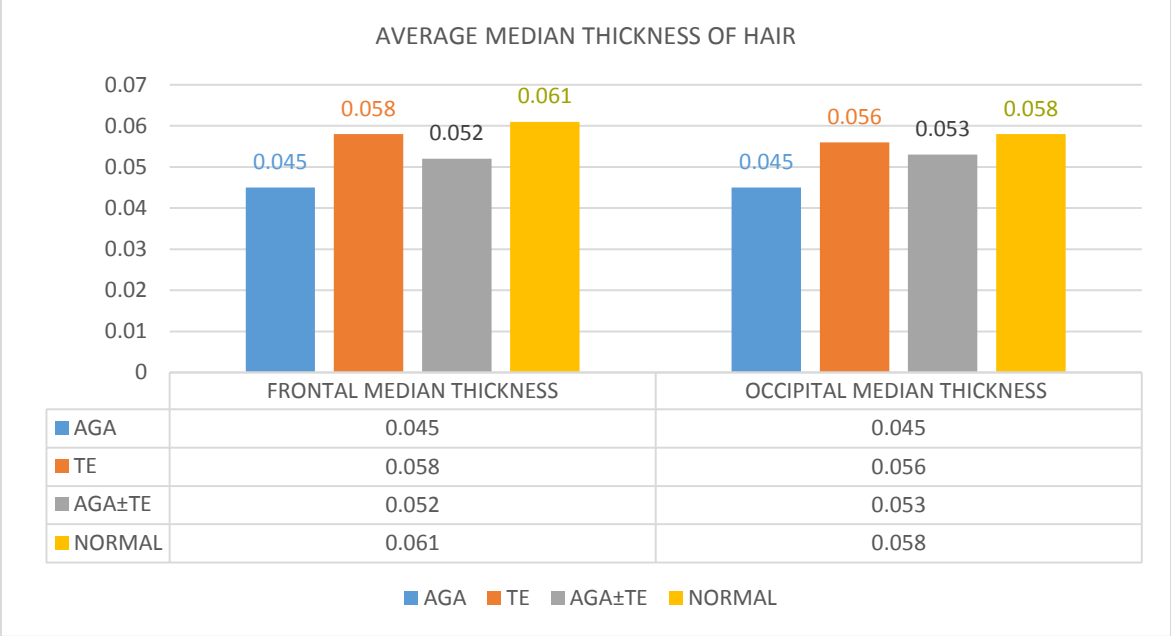
Figure 11: Vellus hair percentage, vellus hair density and single follicular units in non-scarring alopecia

As shown in Figure11 the mean vellus hair percentage and mean vellus hair density was highest in the AGA group followed by AGA+TE group and was least in the telogen effluvium.

The median value of frontal vellus hair percentage was significantly higher in AGA (41.6) than in TE (21.95) and AGA+TE (28.2) (p=0.001). The median value of frontal vellus density was significantly higher in AGA (57.6) than in TE (28.8) (p=0.001). The median value of occipital vellus density was significantly higher in AGA (58.15) than in

TE (32.1) (p=0.024). The single follicular units were higher in AGA than in TE and AGA+TE group but there was no significant difference among the values.

3. *Median thickness of hair in diffuse non-scarring alopecia-*



	FRONTAL MEDIAN THICKNESS	OCCIPITAL MEDIAN THICKNESS
AGA	0.045	0.045
TE	0.058	0.056
AGA±TE	0.052	0.053
NORMAL	0.061	0.058

Figure 12: Median thickness of hair in diffuse non-scarring alopecia.

As shown in Figure 12, the median thickness of hair was lowest in the androgenetic alopecia followed by combination of AGA and TE and then highest in the TE patients. The median value of frontal hair thickness is lower in AGA (0.044 mm) than in TE (0.056 mm) and AGA+TE (0.051 mm), though this was not statistically significant. However, the median value of occipital hair thickness is significantly lower in AGA (0.042 mm) than in TE (0.055 mm) and AGA+TE (0.053 mm) (p=0.000).

4. *Terminal: vellus ratio on trichoscan in diffuse non-scarring alopecia:*

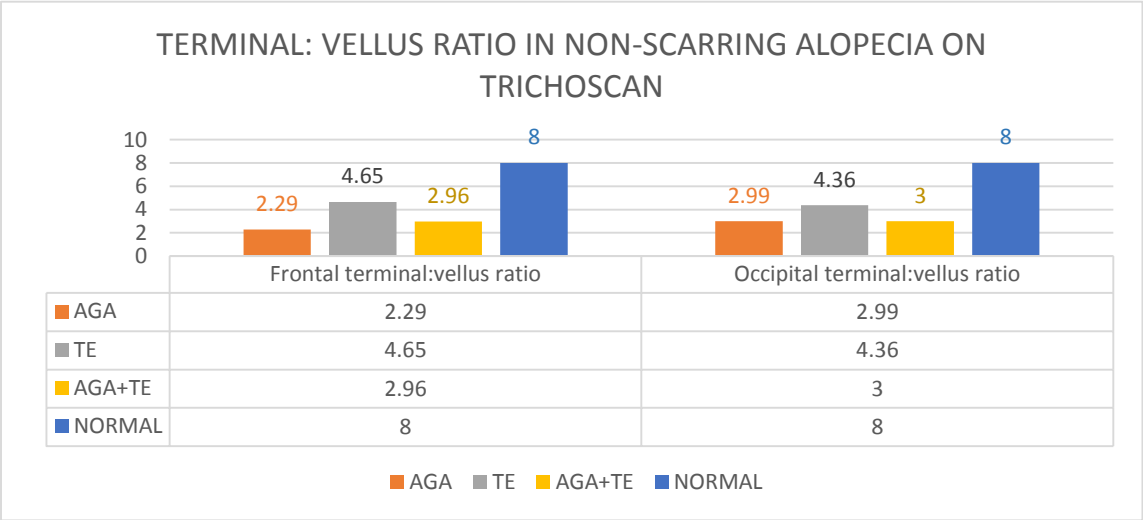


Figure 13: Terminal: Vellus ratio in diffuse non-scarring alopecia

As shown in Figure 13, the terminal: vellus ratio was lowest in the androgenic alopecia followed by AGA+TE group and it was highest in telogen effluvium.

The median value of frontal terminal: vellus ratio was significantly lower in AGA (1.4) than in AGA+TE (2.55) and TE (3.55) (p=0.001). Similarly median value of occipital terminal: vellus ratio was significantly lower in AGA (1.46) than in AGA+TE (2.63) and TE (3.85) (p=0.004).

B. Trichoscopic examination in patchy non-scarring alopecia (alopecia areata)

There were 6 patients (6.25%) with alopecia areata. One patient had alopecia totalis. The mean percentage of scalp involvement in the 5 patients with AA was 6.4%. Trichoscopy was done from all the alopecia patches if there were more than 1 patch in a patient. The trichoscopic findings showed 63.9% patients had numerous yellow dots, 19.4% patients had scanty yellow dots, 44.43% patients had black dots and 63.9% patients had broken

hair. There were 66.66% patients who had short vellus hair and 50% who had short regrowing hair. The trichoscopic findings are shown in Figure 14.

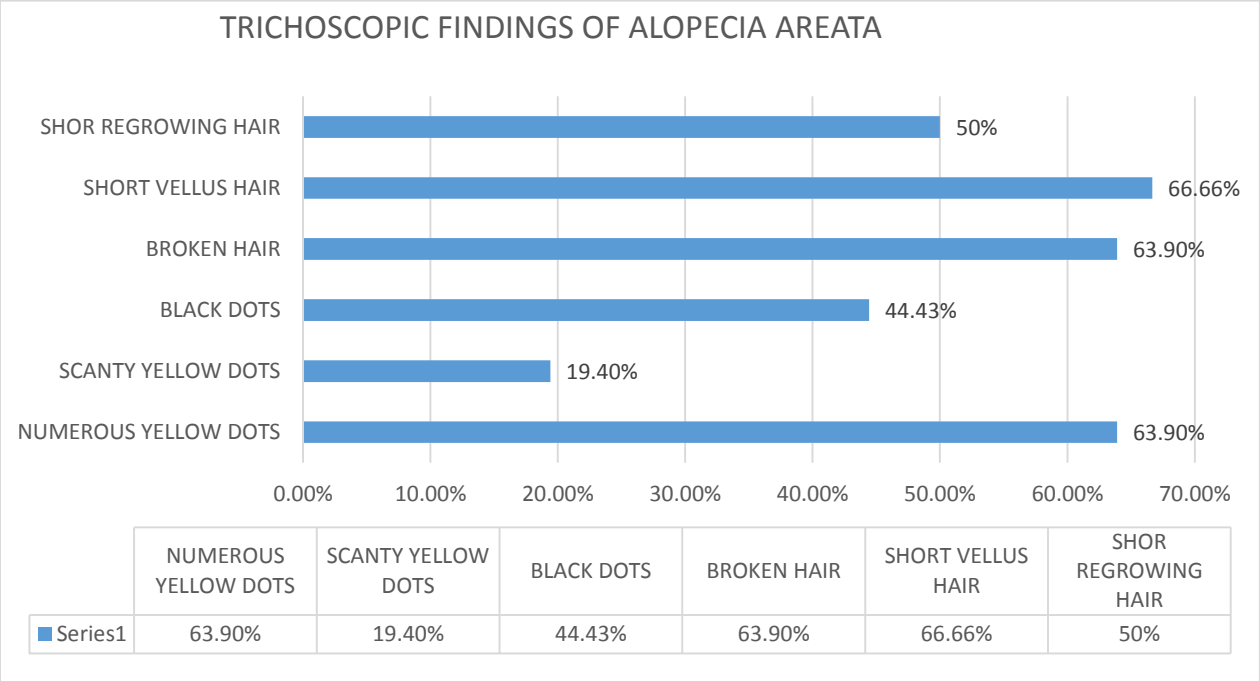


Figure 14: Trichoscopic findings of alopecia areata

C. Trichoscopic Examination In Scarring Alopecia (DLE)

There were 26 patients (27%; 26/96) who had discoid lupus erythematosus, of which 19 patients (19.8%; 19/96) had DLE lesions on the frontal scalp, 22 patients (22.9%; 22/96) had DLE lesions on the temporal scalp and 21 patients (21.9%; 21/96) had DLE lesions on the occipital scalp. There were 11 patients (11.45%) with localized DLE and 15 patients (15.62%) with disseminated DLE. The mean percentage of scalp involvement was 12.12% and it ranged from 1 to 45% involvement of the scalp. Single most representative DLE plaque was selected in each patient for trichoscopy, the results of

which are shown in the Table 16. The commonest trichoscopic findings which were present in more than 80% of patients were blue gray dots and globules (81%), loss of follicular units (84%) and structureless white areas (96%), and the other common findings were hyperkeratotic plugs (69%) and telangiectasia (65%).

Table 16: Trichoscopic findings in DLE

TRICHOSCOPIC FEATURE	NUMBER OF PATIENTS (N=26)	PERCENTAGE
HYPERKERATOTIC PLUGS	17	69%
SCALING	11	44%
NUMEROUS SMALL YELLOW DOTS	3	12%
SCANTY SMALL YELLOW DOTS	6	24%
NUMEROUS LARGE YELLOW DOTS	2	8%
SCANTY LARGE YELLOW DOTS	5	20%
RED DOTS OR GLOBULES	6	27%
TELANGIECTASIA	16	65%
MILKY RED AREAS	4	16%
PERIFOLLICULAR WHITE HALO	2	8%
STRUCTURELESS WHITE AREA	24	96%
LOSS OF FOLICULAR UNITS	21	84%
EXAGGERATED HCP	14	56%
BLUE GRAY DOTS AND GLOBULES	20	81%
BLUE/GRAY/BROWN SPECKLES PIGMENTATION	16	65%

D. Final trichoscopic patterns of non-scarring and scarring alopecia in our study population

After trichoscopic evaluation was done, the distribution of patterns of alopecia found in our study group are shown in Table 17. Telogen effluvium was the commonest pattern of alopecia either solitary (39.58%) or in combination of other types of alopecia.

Table 17: Trichoscopic pattern of alopecia in SLE after trichoscopy

PATTERNS OF ALOPECIA	NUMBER OF PATIENTS n (%)
TE	38 (39.58%)
DLE	16 (16.66%)
AGA	13 (13.54%)
AGA + TE	13 (13.54%)
AGA + DLE	5 (5.20%)
TE + DLE	5 (5.20%)
TE + AA	3 (3.12%)
AA	2 (2.08%)
AGA + AA	1 (1.04%)
TOTAL	***96***

Comparison of yellow dots in non-scarring and scarring alopecia

Yellow dots were highest in alopecia areata (83.33%) followed by androgenetic alopecia (56.4%) and then discoid lupus erythematosus (28%) followed by telogen effluvium (19.1%).

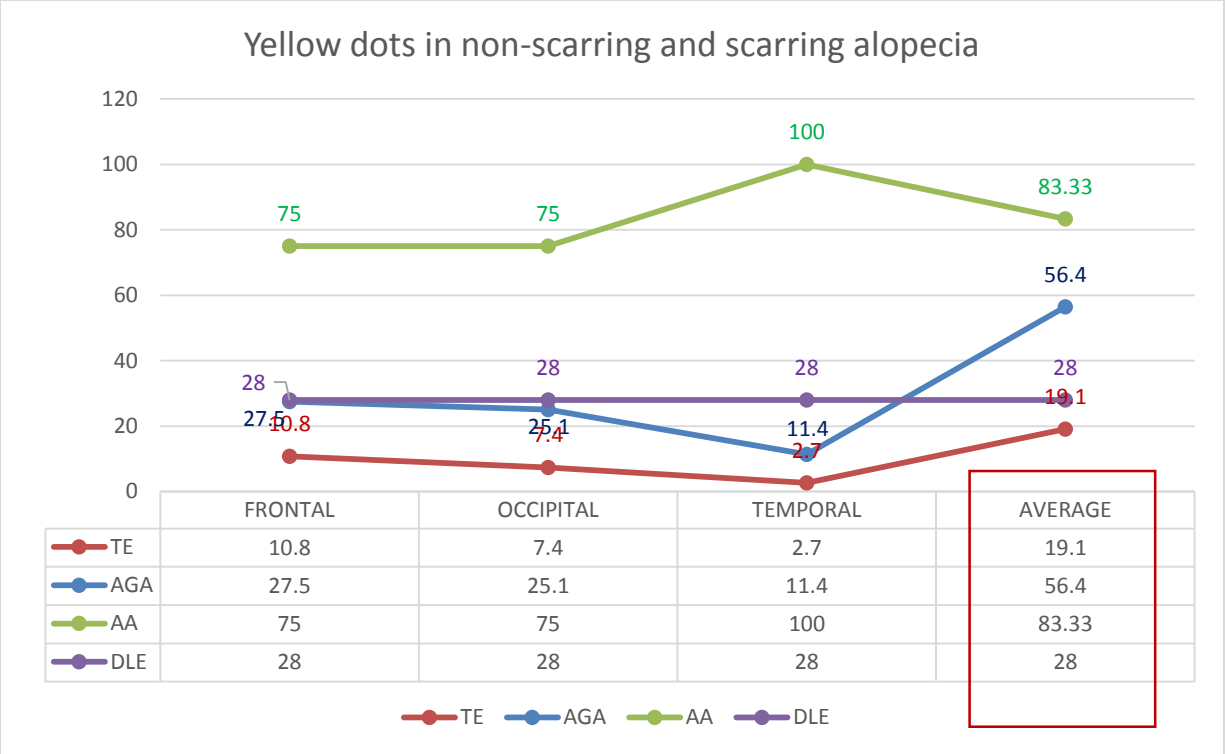


Figure 15: Yellow dots in non-scarring and scarring alopecia

4.2 SLE DISEASE ACTIVITY INDEX (SLEDAI) AND ALOPECIA

1. SLEDAI

The mean SLEDAI score was 11.97 ± 8.495 among all patients. The distribution of SLEDAI score on the basis of severity is given below in Figure 16. The majority of the patients were in the moderate activity (30.2%). There were 29.2% and 18.8% patients with high activity and very high activity respectively.

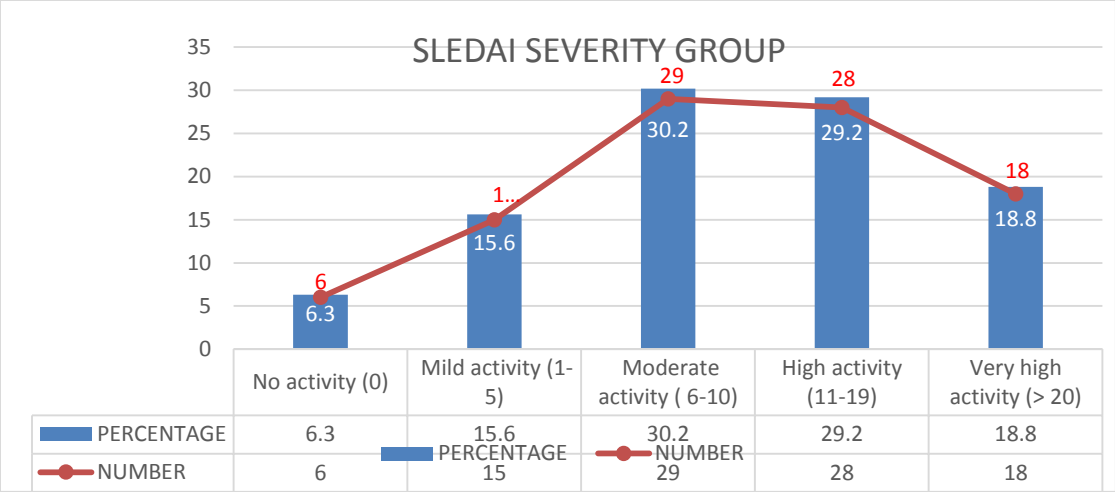


Figure 16: SLEDAI among the patient population

The median SLEDAI scoring in females was 11 and males was 8. There was no statistical significant difference between severity in females and males (p=0.234).

2. Correlation of SLEDAI and clinical manifestations

The amount of hair loss was not statistically significant in relation to SLEDAI scoring (p=0.264). There was no relationship between duration of exacerbation of hair loss and SLEDAI scoring (p=0.63). The distribution of cutaneous lupus erythematosus and SLEDAI is shown in Table 18.

Table 18: The distribution of cutaneous lupus erythematosus and SLEDAI score.

Cutaneous lupus erythematosus	NUMBER (n=96)	SLEDAI scoring Median (interquartile range)
ACLE	28	12 (8.0,18.0)
CCLE	13	9 (3.0, 13.0)
ACLE and SCLE	1	10 (10.0, 10.0)
SCLE and CCLE	1	18 (18.0, 18.0)
ACLE and CCLE	9	19 (14.0, 24.0)
NIL	44	8 (4.0, 13.0)

3. Correlation of patterns of alopecia and SLEDAI

The mean SLEDAI was found to be highest among AGA (15.05) among diffuse non-scarring alopecia followed by telogen effluvium (11.73). The mean SLEDAI among DLE patients was 12.84 and alopecia areata was 8.43. The distribution of SLEDAI among the different types of alopecia was not statistically significant as shown in Table 19.

Table 19: Correlation between patterns of alopecia and SLEDAI score

VARIABLES	SLEDAI score		P VALUE
	Mean ± SD	Median (Interquartile Range)	
Diffuse non-scarring alopecia			0.204
AGA	15.05 ± 8.3	13.5 (8.0, 22.5)	
TE	11.73 ± 8.3	10 (6.0, 16.0)	
AGA+TE	10.46 ± 9.6	7 (4.0, 14.0)	
Absent	10.22 ± 7.9	10 (3.5, 15.75)	
Alopecia Areata			0.265
Present	8.43 ± 4.9	6.0 (6, 12)	
Absent	12.25 ± 8.7	10 (6, 18.5)	
DLE			0.593
Present	12.84 ± 9.2	12 (4.5, 19)	
Absent	11.66 ± 8.2	10 (6, 17)	

Distribution of different patterns of alopecia with SLEDAI is shown in Table 20. There were 63.14% of AGA, 43.47% of TE and 57.69% of DLE patients who had high or very high SLEDAI score.

Table 20: Distribution of alopecia patterns among the severity groups of SLEDAI

SLEDAI SCORING	AGA (n; %)	TE (n, %)	AGA+TE (n, %)	AA (n, %)	DLE (n, %)
No activity (0)	0; 0	2, 4.34	2, 15.38	0, 0	2, 7.69
Mild activity (1-5)	2, 10.52	7, 15.21	2, 15.38	1, 16.66	5, 19.23
Moderate activity (6-10)	5, 26.31	17, 36.95	4, 30.76	3, 50	4, 15.38%
High activity (11-19)	6, 31.57	13, 28.26	2, 15.38	2, 33.33	10, 38.46%
Very high activity (> 20)	6, 31.57	7, 15.21	3, 23.07	0, 0	5, 19.23
TOTAL (N)	**19**	**46**	**13**	**6**	**26**

5. CLINICAL IMAGES

Figure 17: Oral ulcer in ACLE

Figure 18: Subacute cutaneous lupus erythematosus

Figure 19: Female pattern androgenetic alopecia (A,B)

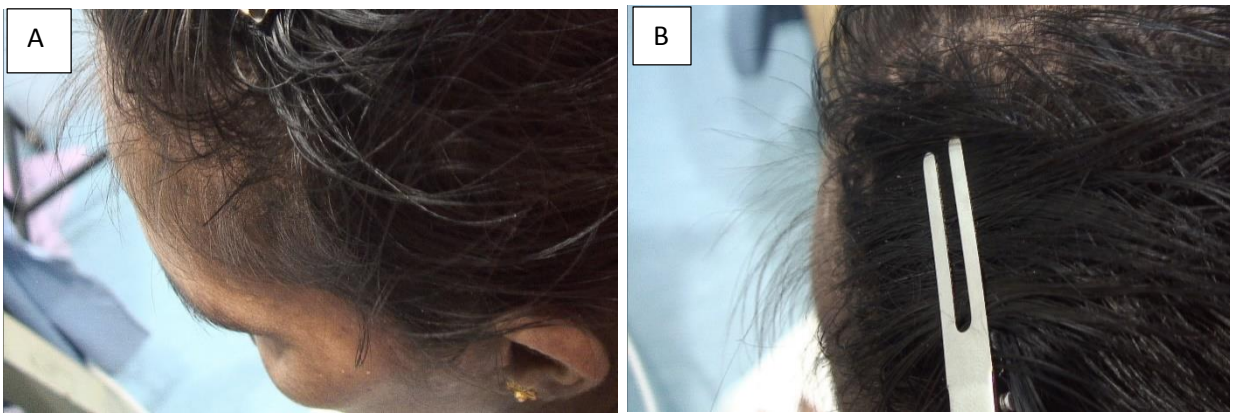


Figure 20: Lupus hair (A,B,C)

Figure 21: Alopecia areata (A-Patchy alopecia areta, B- Alopecia totalis)

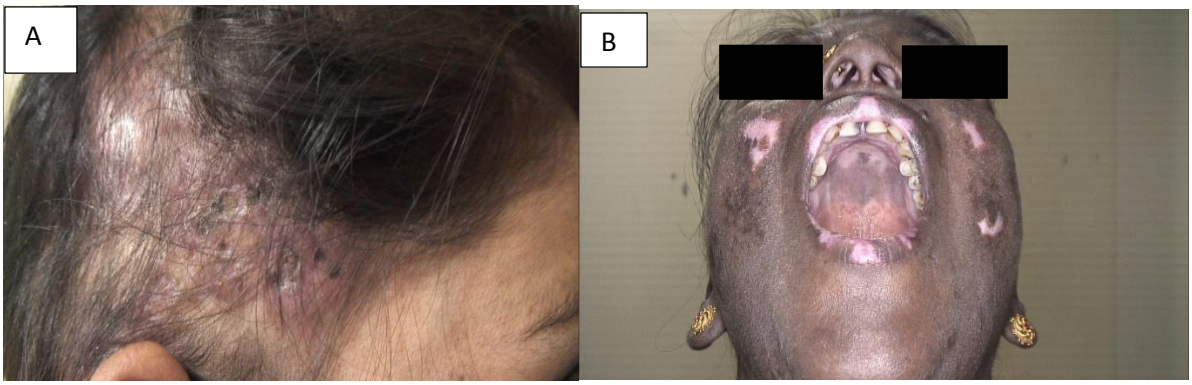


Figure 22: Discoid lupus erythematosus (A) Scalp DLE (B) Mucosal DLE (involving nasal and oral mucosa).

Figure 23: Discoid lupus erythematosus – end stage (A, B)

6. TRICHOSCOPIC PHOTOGRAPHS

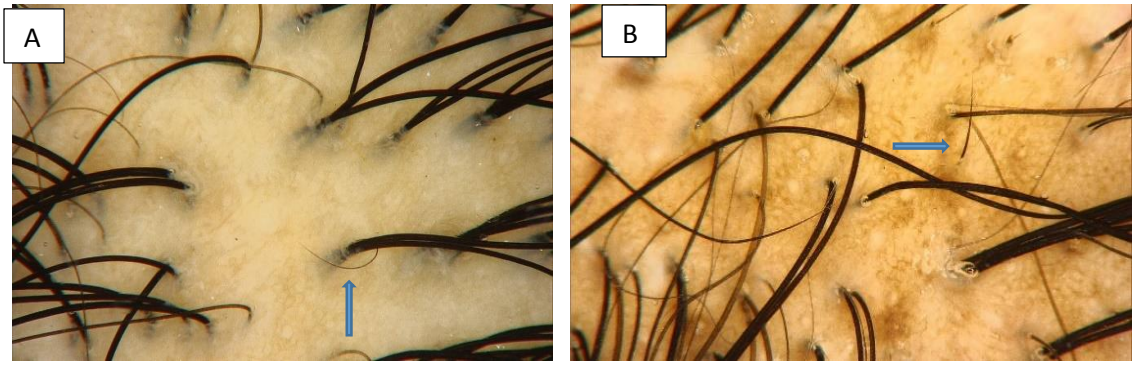


Figure 24: Short regrowing hair- blue arrow (20X) (A, B)

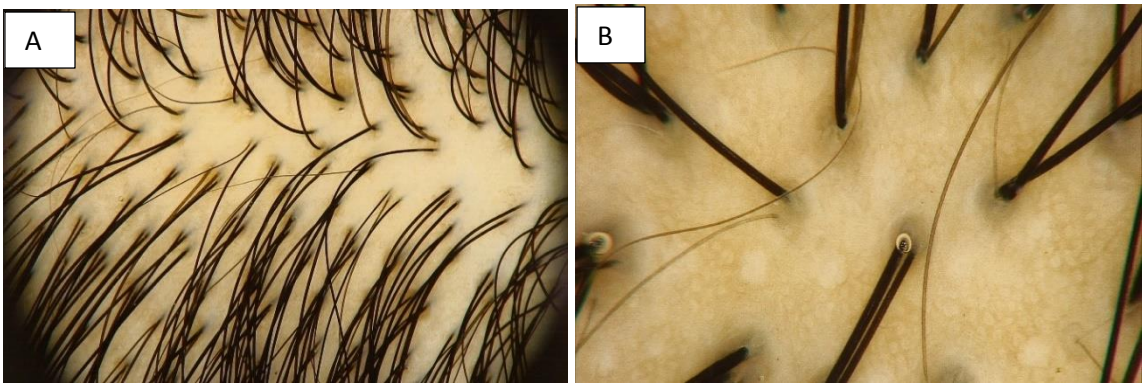


Figure 25: Peripilar sign (A) 20X (B) 70X

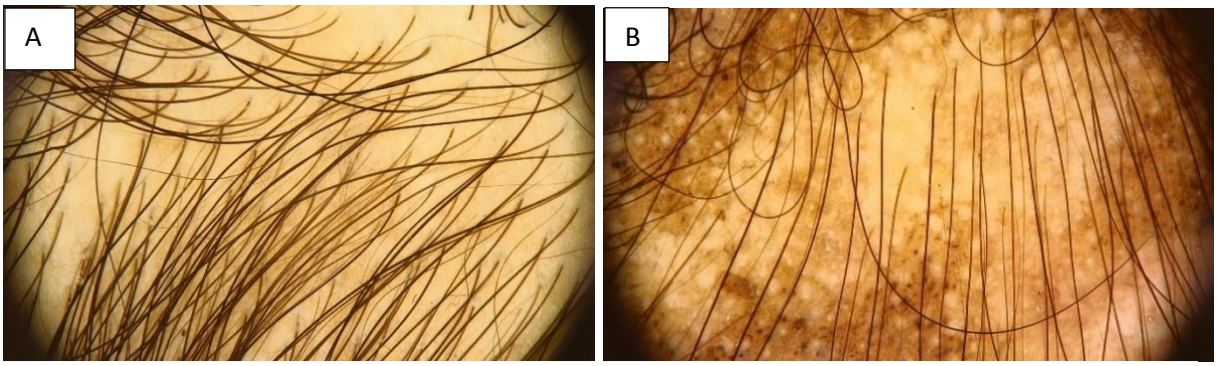


Figure 26: Predominant single follicular units (20X) (A,B)

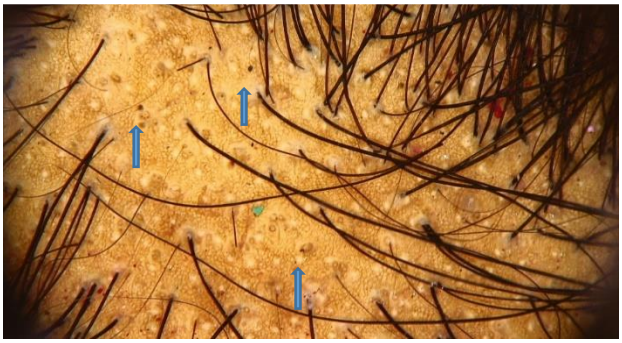


Figure 27: Multiple black dots (blue arrows) and broken hair in alopecia areata- (20X)

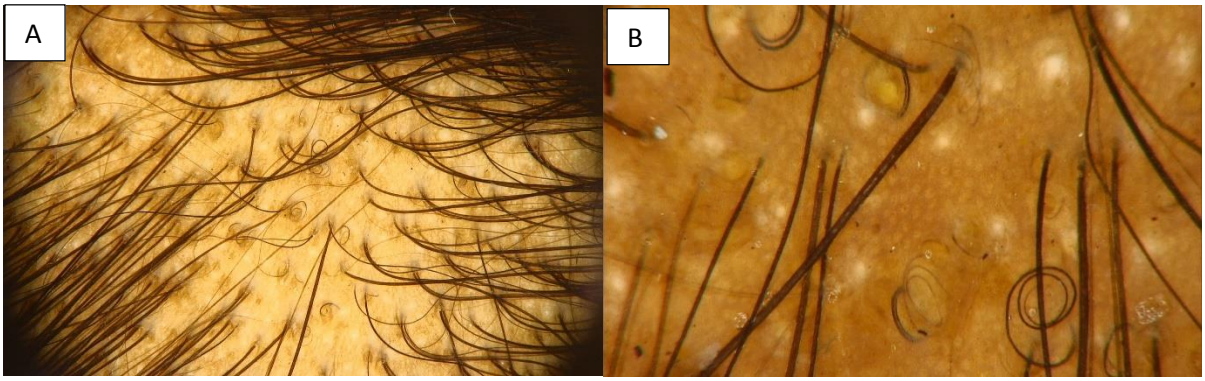


Figure 28: Circle hair (pigtail hair) in alopecia areata – (A) at 20X, (B) at 70X

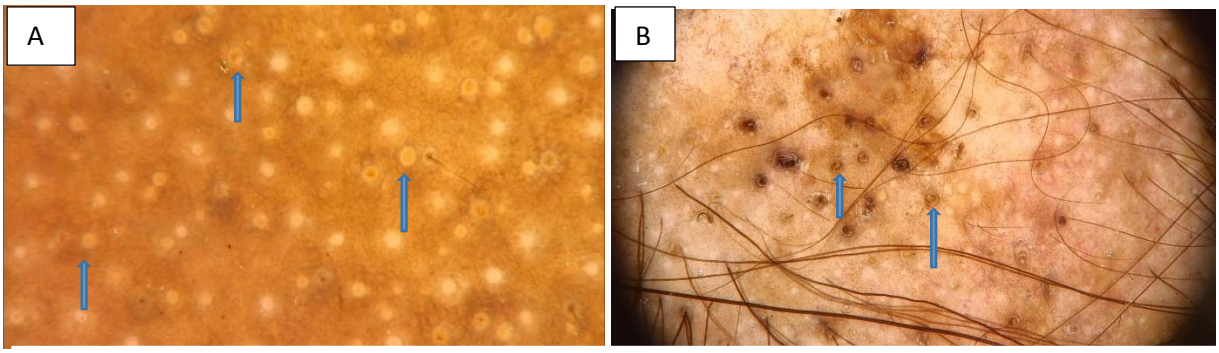


Figure 29: Numerous yellow dots in alopecia areata (A), Scanty yellow dots in DLE (B)

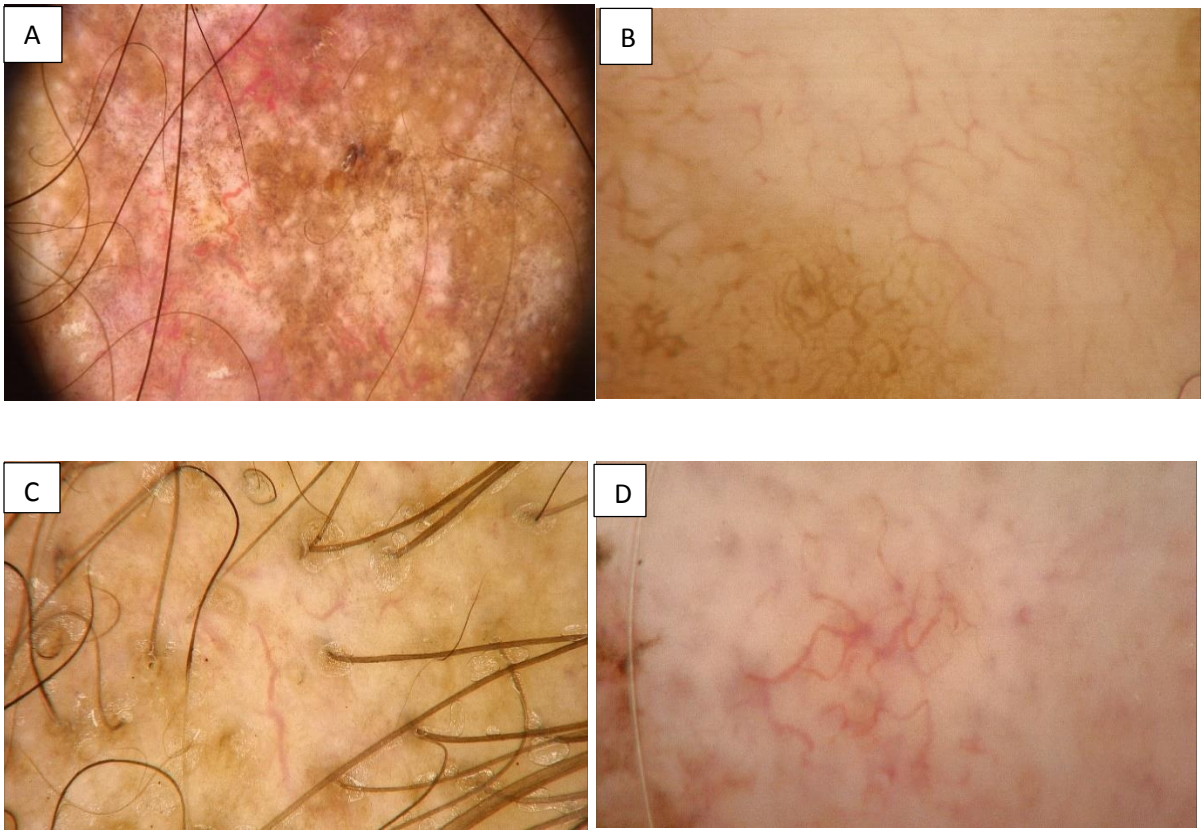


Figure 30 Telangiectasia in DLE (A and C. at 20X, B and D. at 70X)

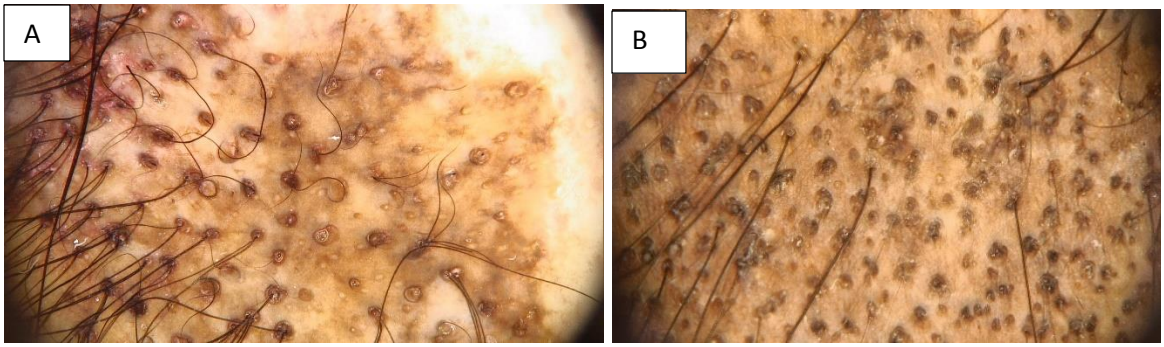


Figure 31: Hyperkeratotic plugs in DLE (20X) (A,B)

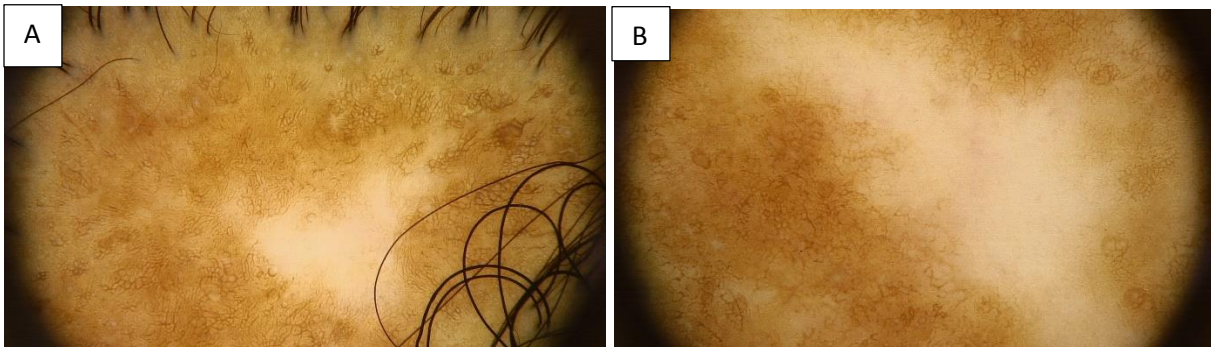


Figure 32: Large structure less white areas (A,B)

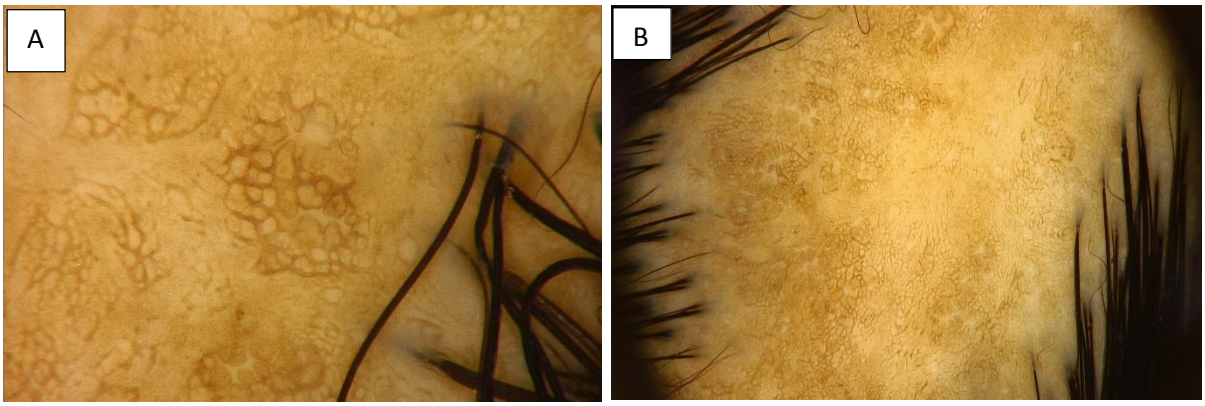


Figure 33: Exaggerated honeycomb pigment network (A. at 70X, B. at 20X)

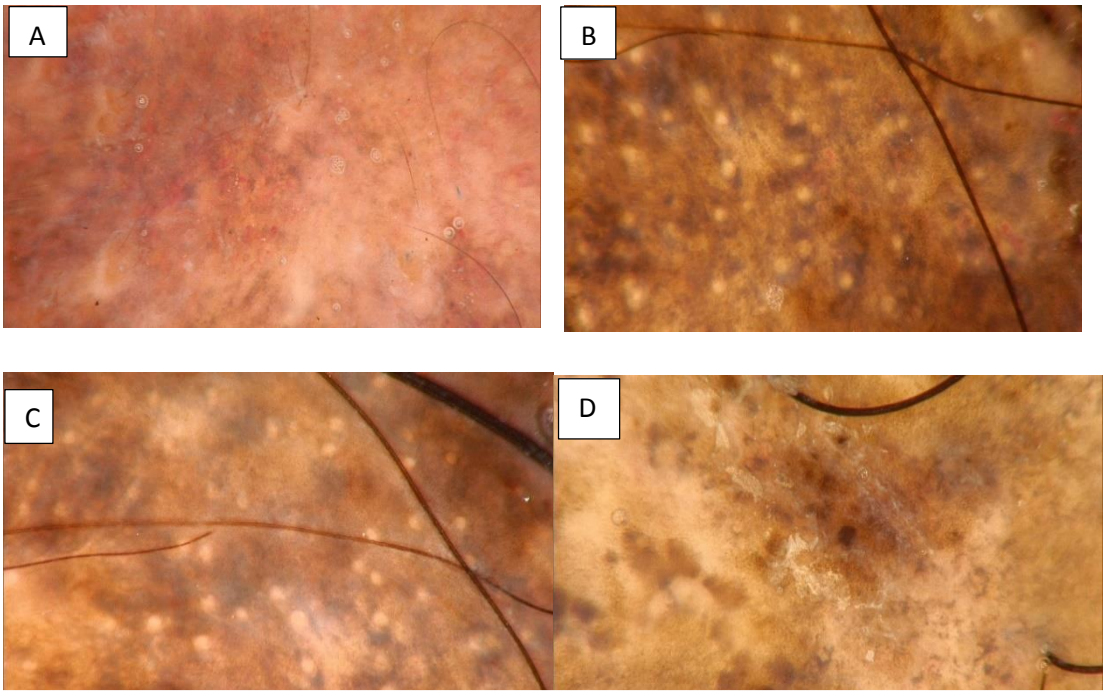


Figure 34: Blue gray globules and blue gray/brown speckled pattern

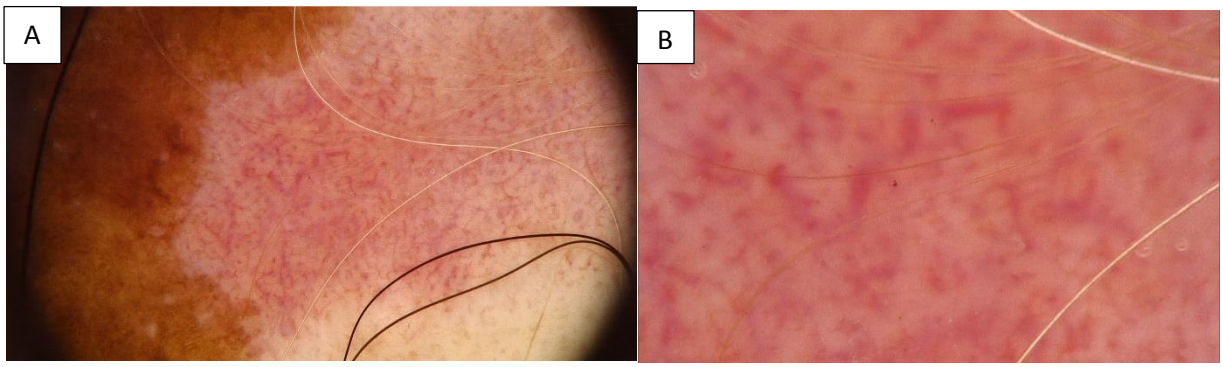


Figure 35: Red dots and globules (A. at 20X, B. at 70X)

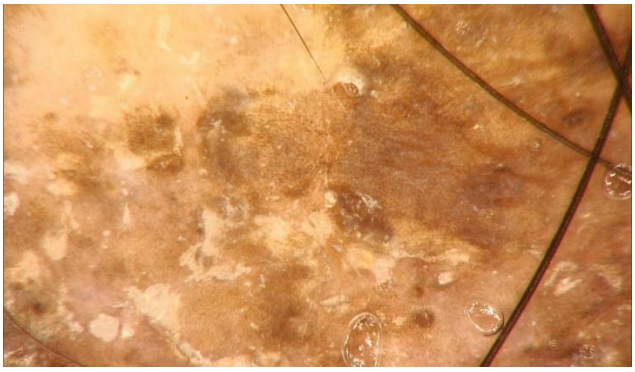


Figure 36: Scales (on wet trichoscopy) at 20X

DISCUSSION

Systemic lupus erythematosus (SLE) is a multisystem connective tissue disease involving most commonly the skin, joints and vasculature and is associated with immunological abnormalities. They usually present with fever, photosensitive skin rashes and arthritis. Systemic involvement of the renal system is more commonly seen, however pulmonary, cardiac and neurological involvement can also occur.

Hair loss in SLE is common and is seen in around 20-60% of the patients (4). The type of hair loss can be either scarring or non-scarring. The non-scarring type of hair loss seen among the patients include androgenetic alopecia, telogen effluvium and anagen effluvium. Scarring alopecia is usually due to the chronic cutaneous lupus erythematosus (discoid lupus erythematosus).

Diffuse non-scarring alopecia is the most common non-specific skin manifestation of SLE and occurs in more than 60% of cases either transiently or during increased disease activity. Alternatively, the alopecia can be chronic which can lead to coarse, dry and fragile hair along the peripheral hairline known as ‘lupus hair’.

In a study by Werth *et.al.* (61), alopecia areata is also more often reported, in approximately 10% of patients with SLE in contrast to 2% overall risk of alopecia areata in general as observed in Miteva *et.al.* in 2015 (114).

Permanent scarring alopecia due to the discoid lupus erythematosus, is a LE- specific clinical manifestation (6–8).

Various criteria have been evolved for the diagnosis of SLE. The recently introduced SLICC criteria for the diagnosis of SLE, included alopecia as one of the clinical criterion used for the diagnosis of SLE, however the earlier ACR criteria did not include the same. Alopecia also forms a part of the SLEDAI (systemic lupus erythematosus disease activity index) scoring system for measurement of disease activity and has a score of one point out of 105 points.

1. **Clinical profile of patients**

Ninety six patients with SLE were included in our cross sectional observational study on patterns of alopecia. All the patients included with SLE complained of hair loss at some point of time during the disease.

The majority of our patients were in the 20-39 years age group. The mean age of onset of disease in our study was 27.99± 9.66 in females and 26.54 ±9.48 in males (average 27.26 years). This was higher than the age of onset in the study conducted by Malaviya *et.al.* in 1997 in India which was 24.5 years (23). But this was much lower than the age of onset seen in the study by Lim *et.al.* in 2014, where it was 40.5 years (22).

The sex ratio in our study was 6:1 (F: M). This was in concordance with the study conducted by Feldman *et.al.* during 2000-2004 (19) but much lower than the study by Malaviya *et.al.* (23). The mean duration of disease in our study was 4.28 years. The longest duration of SLE in our study was 18 years.

Table 21: Comparison of the age of onset and sex distribution in various studies

Parameter	**Present study**	**Feldman *et.al.*** (19)	**Garris *et.al.*** (24)	**Sadana *et.al.*** (25)	**Soto *et.al.*** (26)	**Jarukitsopa *et.al.*** (27)	**Yun *et.al.*** (4)	**Malviya *et.al.*** (21)
Total number of patients in study	96	34,339	13,348	20	187	45	122	1366
Type of study	C	R	R	R+P	R	R	C	R
Year of study	2016-17	2000–2004	2003-2007	1955-1962	1982-2002	1993 to 2005	2004	1997
Ethnicity	Indian	Low-income U.S. Medicaid Population	US Medicare population	Indian	Mexico	White US population	Korean	Indian
Mean age Age range	Mean age- 27.26 years	Mean age- NA Age range -18 – 65	Mean age -61 Age range -NA	Mean age- NA Age of onset range- 10-53	Mean age- 31 Age range- F- 10-75 M- 7-65	Mean age- 42 Age range- NA	Mean age- 32.7 Age range- 13-71	Mean age- 24.5 Age range- 4-75
Female: Male Ratio	6:1	6:1	6:1	13:7	4.2:1	10:1	12.6:1	11:1

(R- retrospective study, P- prospective, C- crossectional study)

2. Comparison of patterns of alopecia in SLE with various studies

Alopecia was observed in 20-60% of SLE as observed by Yun *et.al.* in Korean population. On literature search, only one study describing the patterns of alopecia in SLE by Yun *et.al.* in Korean population, was available. The other studies were mostly on cutaneous and systemic manifestations in SLE patients. These studies described hair loss as a common manifestation but did not describe the details of the patterns of different type of non-scarring alopecia (20,33–37). In SLE, both non-scarring and scarring alopecia can be seen. The diffuse non-scarring alopecia is a non-specific lupus cutaneous manifestation and is the most common pattern of hair loss. This pattern also forms one of the clinical diagnostic criteria in SLICC criteria. The comparison of patterns of alopecia in our study with various studies is described in Table 22.

Table 22: Patterns of alopecia in SLE- comparison with other studies

Variable	**Present study**	**Yun *et.al.* study**	**P. Salphale *et.al.***	**Kapadia *et.al.***
Population	Indian	Korean	South India	Pakistan
Study period	2016-17	2004	2011	1996
Number of patients	96	122	93	40
Diffuse non-scarring alopecia: 1. TE 2. AGA 3. TE+AGA 4. AE 5. Lupus hair	 46.9% 20.80% 13.5% 0% 14.6%	 65.1% 10.5% Not mentioned 12.8% 15.1%	 73.3% Not mentioned Not mentioned Not mentioned 25%	Diffuse non-scarring alopecia- 82.5% Not specified Not specified Not specified 12.5%
Patchy non-scarring alopecia (AA)	6.25%	15.1%	1.7%	NA
Scarring alopecia (DLE)	27.08%	7%	16.1%	15%

In a report by Kapadia *et.al.* (35) on cutaneous manifestation of SLE (n=40), they found diffuse non-scarring alopecia in 82.5%, lupus hair in 12.5%, and DLE in 15%. They did not describe the subtypes of diffuse non-scarring alopecia in SLE. In the study by Yun *et.al.*, on the patterns of hair loss in SLE, reported telogen effluvium in 65.1%, female pattern hair loss in 10.5%, anagen effluvium in 12.8% and 'lupus hair' in 15.1% of patients. The diagnosis of alopecia in this study was based on history and clinical examination. The prospective cross-sectional study conducted by P. Salphale *et.al.* (37) in 2007-08 from south India aimed to study the severity of skin and mucosal involvement in SLE using the Cutaneous Lupus Erythematosus Disease Area and Severity Index (CLASI) activity and damage score, with 93 patients of SLE having cutaneous manifestaions. This study showed non-scarring alopecia in 64.5% patients and DLE in 16.1% patients. Among the non-scarring alopecia, there were 73.3% patients with telogen effluvium, 25% patients with lupus hair and 1.7% patients with alopecia areata.

In our study, 95.8% patients gave history of hair loss at presentation and 14.5% patients had history of hair loss even before the diagnosis of SLE. The mean duration of hair loss was 3.30 years, and the maximum duration was 25 years. There were 35.4% patients who gave history of hair loss of more than 200 hairs per day.

We had 78 patients (81.25%) with diffuse non-scarring alopecia, among them 46 patients (47.91%) had telogen effluvium, 19 patients (19.79%) had androgenetic alopecia and 13 patients (13.50%) had combination of telogen effluvium and androgenetic alopecia. There were 14.6% patients who had lupus hair. Only 6.25% of patients in our study had

alopecia areata in comparison to the study by Yun *et.al.* who reported 15.1% patients with alopecia areata. In our study the mean age of onset of AGA was 29 years and AGA+TE was 30.38 years. Also, we had relatively more number of patients with AGA. This may be probably due to the earlier detection of miniaturization of scalp hair that was picked up with the use of trichoscope. Clinically evident AGA among patients with diffuse nonscarring alopecia, was seen in only 14 patients (14.58%) in our study.

Jang *et.al.* in 2013 (55), retrospectively studied the age of onset and severity of AGA in both genders among Korean patients, and reported that the mean age of onset of patients with AGA in 2010 was 31.6 years. This study also compared the mean age of onset of AGA from 2006 to 2010 and found that the age of onset of AGA in 2010 is earlier (31.6 years) in comparison to 34.1 years in 2006. Erdogan *et.al.* postulated that there is an increased oxidative stress in patients with early-onset AGA (56).

The findings seen in our patients with AGA were corroborated by trichoscan. The present study is the only study that has categorised alopecia in SLE with the aid of a trichoscope. We also found much higher number of patients with DLE i.e. 27.08% in comparison of only 7% of DLE patients in the study by Yun *et.al*. None of the patients in our study had anagen effluvium.

3. **Trichoscopy findings**

A. Comparison of trichoscopic features and trichoscan measurements in diffuse non-scarring alopecia with various studies

The Table 23 compares the trichoscopic findings of AGA and TE in our study.

Table 23: Comparison of trichoscopic findings in diffuse non-scarring alopecia.

Variable	**Present study**			
Variable	**AGA**		**TE**	
Scalp region	Frontal (n=29)	Occipital (n=16)	Frontal (n=46)	Occipital (n=54)
Scanty yellow dots	17.20%	18.80%	6.50%	7.40%
Numerous yellow dots	10.30%	6.30%	4.30%	0%
Peripilar sign	20.70%	25%	21.70%	20.40%
Short regrowing hair	44.80%	31.30%	45.70%	44.40%

We compared trichoscopic findings of AGA and TE with each other. The percentage of patients with AGA having yellow dots were higher (56.4%) than patients with telogen effluvium (19.1%). The mean yellow dots in AGA in the study by Rakowska *et.al.* was 8.86 ± 4.8 in frontal scalp and 1.59 ± 2.0 in occipital scalp per 4 fields of vision at the 70-fold magnification. The number of yellow dots in each patient in our study population was much lower than the study by Rakowska *et.al.* in the Polish population. Hence, we would need more trichoscopic studies on AGA in Indian population to compare these findings.

Short regrowing hair were found more in the telogen effluvium (52.56% of patients) than androgenetic alopecia (40.6% of patients) though this was not statistically significant

(p=0.275). The study by Rakowska et.al also reported more short regrowing hair in TE than AGA. Peripilar sign was found to be more or less equal in androgenetic alopecia (18.10%) and telogen effluvium (17.63%) in our study. This was in contrast to the study by Rakowska *et.al.* (112) in which peripilar sign was found significantly more in AGA as compared to the TE. More studies are required in Indian population to comment on this feature.

For diffuse non-scarring alopecia, trichoscan was carried out by an in-built software in the videodermoscopes which allows to carry out measurements of structures visualized in magnified photographs and provides results in real scale. In 2009, Rakowska *et.al.* (112), in Polish population also conducted trichoscan for diffuse non-scarring alopecia. This study was conducted on patients with either telogen effluvium or androgenetic alopecia diagnosed on the basis of clinical examination and histopathology. As our study involved trichoscopy of scalp for the diagnosis of alopecia, we were able to detect early androgenetic alopecia without clinically evident AGA. There was also combination of diffuse non-scarring alopecia in our study as patients had combination patterns which were detected on trichoscopy which might have been difficult to diagnosis only on the basis of history and clinical examination. The study by Rakowska *et.al.* reported, a ratio of *Terminal verses vellus hair ratio* of 8:1 among healthy individuals. The ratio of less or equal to 4:1 is considered as FAGA. In our study the patients with AGA had terminal verses vellus ratio less than 4:1; it was 2.29 in the frontal scalp and 2.99 in the occipital scalp. Patients with TE showed more than 4:1 ratio in both frontal and occipital scalp; it

was 4.65 in the frontal scalp and 4.36 in the occipital scalp. There are no studies describing the normal values of trichoscan in Indian population. The comparison of trichoscan measurements with the study by Rakowska et.al. is shown in Table 24.

Table 24: Comparison of trichoscan measurements

Variable	**Present study (n=96)**				**Rakowska *et.al.* (n=131)**			
Diffuse non-scarring alopecia	**TE (n=46)**		**AGA(n=19)**		**TE (n=33)**		**AGA (n=59)**	
Scalp region F- Fontal O- Occipital	**F**	**O**	**F**	**O**	**F**	**O**	**F**	**O**
Vellus hair (%)	**23.2**	**22.44**	**39.81**	**39.06**	**10.4**	**NA**	**20.9**	**NA**
Single follicular units (%)	**47.16**	**45.61**	**49.44**	**46.78**	**39**	**31**	**65.2**	**36.8**
Mean hair thickness (mm)	**0.058**	**0.056**	**0.045**	**0.045**	**0.056**	**0.053**	**0.047**	**0.052**

In the study by Rakowska *et.al.* in 2009, the *mean percentage of vellus hair* (< 0.03 mm) in frontal scalp was 20.9 ± 12% in androgenic alopecia, 10.4 ± 3.9% in telogen effluvium and 6.15 ± 4.6% in healthy individuals. In comparison, our study also showed higher mean percentage of vellus hair in AGA (39.81±16.70% in frontal and 39.06±18.02% in occipital) than in TE (23.2±10.95% in frontal and 22.44±8.79% in occipital). We found slightly lesser vellus hair percentage in occipital scalp than frontal scalp as shown in Table 24.

In the study by Rakowska *et.al.,* the *mean percentage of single-hair pilosebaceous units* in healthy individuals was 27.3 ± 13 and 22.6 ± 12.6 in frontal and occipital scalp respectively. In FAGA, it was 65.2 ± 19.9 in the frontal scalp, and 36.8 ± 18.6 in the occipital scalp. In TE, it was 39.0 ± 13.4 and 31 ± 23 in the frontal and occipital scalp respectively. In our study, more single follicular units (SFU) were seen in AGA when compared with TE. The mean percentage of single follicular units was 49.44 and 46.78 in frontal scalp and occipital scalp in AGA, and 47.16 and 45.61 in frontal and occipital scalp in TE. Single follicular units were more in frontal than in occipital scalp in both AGA and TE.

Rakowska *et.al.*, reported, the *average hair thickness* in frontal area and occiput as 0.061 ± 0.008 mm and 0.058 ± 0.007 mm respectively in healthy controls; as 0.047 ± 0.007 mm and 0.052 ± 0.008 mm respectively in androgenic alopecia; and it was 0.056 ± 0.007 mm and 0.053 ± 0.009 mm respectively in TE. In our study the average hair thickness was 0.058mm in frontal and 0.056 in occipital in TE. It was 0.045mm in both frontal and occipital scalp in AGA. Therefore, similar to the study by Rakowska *et.al*., the average hair thickness was less in AGA in comparison to TE in our study.

B. Comparison of trichoscopic features of alopecia areata in various studies

The comparison of trichoscopic findings with other studies is shown in Table 25.

Table 25: Comparison of trichoscopic features of alopecia areata in various studies

Variable	**Present study**	**Inui *et.al.*** (100)	**Chiramel *et.al.*** (101)	**Ankad *et.al.*** (93)	**Naveen *et.al.*** (75)	**Dincy *et.al.*** (102)	**Thappa *et.al.*** (103)
Total patients	6	300	24	50	75	57	66
Place	India	Japan	India	India	India	South India	South india
Yellow dots	Numerous- 63.9% Scanty- 19.4%	63.7%	87.5%	50%	57.33%	42%	81.8%
Black dots	44.43%	44.3%	79.2%	20%	84%	75%	66.6%
Broken hair	63.9%	45.7%	70.8%	30%	37.33%	67%	55.4%
Short vellus hair	66.66%	72.7%	50%	10%	68%	56%	40.9%
Shot regrowing hair	50%	31.7%	Not calculated	Not calculated	18.67%	33%	12.1%

The hallmark trichoscopic features of alopecia areata are regularly distributed yellow dots, micro–exclamation mark hairs, tapered hairs, black dots (formerly called cadaverous hairs), broken hairs, clustered short vellus hairs (shorter than 10 mm) and regrowing upright or regrowing coiled hairs in the areas of hair loss (90,97). Trichoscopy of alopecia areata may differ depending on disease activity, severity, and duration (59,100).

There were 6 patients (6.25%) with alopecia areata in our study. The trichoscopic findings were as follows: 63.9% patients had numerous yellow dots, 19.4% patients had scanty yellow dots, 44.43% patients had black dots and 63.9% patients had broken hairs. These findings were similar to the study conducted by Inui *et.al.* (100). Though the number of patients in our study group with alopecia areata were very less (6 patients) in comparison to 300 patients in the study by Inui *et.al*.

C. Comparison of trichoscopic features of DLE with various studies

In our study there were 26 patients (27%; 26/96) who had discoid lupus erythematosus, of which 19 patients (19.8%; 19/96) had DLE lesions on the frontal scalp, 22 patients (22.9%; 22/96) had DLE lesions on the temporal scalp and 21 patients (21.9%; 21/96) had DLE lesions on the occipital scalp. The mean scalp involvement was 12.12% and ranged from 1- 45% involvement of the scalp. The comparison of trichoscopic findings with other studies is shown in the Table 26.

Table 26: Comparison of trichoscopic features of DLE in various studies.

Variable	Present study	Lallas *et.al.* (81)	Duque *et.al.* (79)	Thakur *et.al.* (42)	Hashem *et.al.* (77)
Total patients	26	55	5	10	5
Structureless white area	96%	36.40%	100%	100%	40%
Loss of follicular units	84%	NA	80%	100%	NS
Blue gray dots and globules	81%	NA	40%	20%	NS
Hyperkeratotic plugs	69%	67.30%	100%	90%	100%
Blue/gray/brown speckled pigmentation	65%	NA	NA	NA	20%
Telangiectasias	65%	52.70%	100%	80%	80%
Exaggerated HCP	56%	43.60%	40%	70%	NA
Scaling	44%	49.10%	0%	80%	40%
Red dots or globules	27%	36.40%	NA	NA	40%
Scanty small yellow dots	24%	NA	NA	NA	NA
Scanty large yellow dots	20%	NA	NA	NA	NA
Milky red areas	16%	NA	NA	NA	NA
Numerous small yellow dots	12%	NA	NA	70%	20%
Perifollicular white halo	8%	69.10%	NA	NA	NA
Numerous large yellow dots	8%	NA	NA	NA	20%

(NA-not available, NS- not specified)

Trichoscopic features of discoid lupus erythematosus described in active (early) lesions are thick arborizing vessels, large yellow dots (follicular keratotic plugs), fine interfollicular scaling, scattered brown discoloration, red dots and blue-gray dots (on dark or sun-exposed skin). While inactive lesions show loss of follicular openings, white areas, pink areas, arborizing vessels and yellow dots containing thin spider vessels (in prefibrotic lesions) (78,81,90,104). The study by Duque *et.al.* in 2010 (79), reported the blue gray dots in DLE are seen in a speckled pattern where as in LPP they have a target pattern. Duque *et.al.* in 2010 (79), and Lanuti *et.al.* in 2012 (76) and Ankad *et.al.* in 2013 (80) also reported the speckled pattern of blue gray dots in DLE. The study Lallas *et.al.* in 2013 reported, perifollicular whitish halo, follicular keratotic plugs and telangiectasias were the most common dermoscopic criteria with a frequency of 69.1%, 67.3% and 52.7%, respectively (81). In 2009, Tosti *et.al.* first described the follicular red dots in active DLE (82) and reported 38% of the patients with red dots. In the study by Lallas *et.al.* (81) red dots were found in 36.5% of patients.

Thakur *et.al.* from North east India in 2015 studied DLE trichoscopic features in 10 patients and found structureless white patches, loss of follicular units in 100% and epidermal atrophy and perifollicular erythema in 100%; hyperkeratotic plugs in 90%; telangiectasias and perifollicular scaling in 80%; yellow dots and brown discoloration in 70%; black dots and blue gray dots in 20% of the patients (42).

In our study the trichoscopic findings in DLE were structureless white patches in 96%, loss of follicular units in 84%, blue gray dots in 81%, hyperkeratotic plugs in 69%, telangiectasias in 65%, blue/brown speckled pigmentation in 65%, exaggerated honey

comb pigmentation in 56%, scaling in 44%, red dots in 27%, milky red areas in 16% patients perifollicular white halos in 8%. Yellow dots were found as numerous small, scanty small, numerous large, scanty large in 12%, 24%, 8%, 20% patients respectively.

We had very less perifollicular white halos in comparison to structureless white areas which were much higher. This could be because most of our patients had inactive and long standing DLE lesions.

D. ***Comparison of yellow dots in non-scarring and scarring alopecia in various studies*** Yellow dots in comparison of other previous studies is shown in Table 27.

Table 27: Comparison of yellow dots in different patterns of alopecia.

Variable	**Present study**	**Chiramel *et.al.*** (101)
Alopecia areata	83.33%	87.5%
AGA	56.4%	FAGA-44.5% AGA- 100%
Telogen effluvium	19.1%	30%
DLE	28%	Not mentioned

Yellow dots were observed in AGA, TE, AA and DLE. Chiramel *et.al.* 2016 (101) from north India, reported yellow dots in 87.5% of alopecia areata, 44.5% of female pattern hair loss, and 30% of telogen effluvium. They did not mention yellow dots in DLE. Hashem *et.al.* (77) reported 20% of DLE cases with yellow dots whereas Thakur *et.al.* (42) reported 70% of DLE cases with yellow dots.

In our study, yellow dots were highest in alopecia areata (83.33%) followed by androgenetic alopecia (56.4%) and then discoid lupus erythematosus (28%) followed in last by telogen effluvium (19.1%).

4. Correlation with disease activity

In 2012 Petri *et.al.*, proposed SLICC criteria (Systemic Lupus International Collaborating Clinics classification criteria for systemic lupus erythematosus) (31) (Annexure 3) for the diagnosis of SLE. Non-scarring alopecia was introduced in the SLICC criteria as well as in SLEDAI scoring for the measurement of disease acivity.

In 2001, RD ZecÏevicÂ *et.al.* studied the correlation between skin lesions and disease activity and found that the disease activity (SLEDAI) score was higher in patients with LE non-specific lesions than patients with LE specific lesions (7.34 verses 17.25 respectively) (115). The alopecia in SLE is found to be associated with disease activity by Wysenbeek *et.al.* in 1991 (5). Though in the study by Yun *et.al.* (4), the relation between SLEDAI and alopecia was found to be insignificant.

In our study the mean SLEDAI score was 11.97 ± 8.495 among all patients. The distribution of patients in the severity groups of SLEDAI score is given in Table 20. The majority of the patients showed moderate activity (30.2%). Similar to the study by Yun *et.al.*, there was no significant correlation between alopecia and SLEDAI. Though non-scarring alopecia was included in SLICC criteria, the types and patterns of alopecia and its correlation to SLEDAI have not been extensively studied so far and thus this study will enlighten our knowledge in this regard.

CONCLUSIONS

1. The majority of patients in this study were in the 20-39 age group and the male to female sex ratio was 1: 6.6.
2. The mean age of onset of SLE in females and males were 28 years and 27 years respectively.
3. Majority of the patients with diffuse non-scarring alopecia had onset of hair loss between 18-30 years.
4. The majority of the study population had diffuse non-scaring alopecia (81.25%) followed by DLE in 27.08%, lupus hair in 14.6% and alopecia areata in 6.25%.
5. Among diffuse non-scarring alopecia, majority of the patients had telogen effluvium 47.91% followed by androgenetic alopecia in 19.79% and combination of both in 13.5%.
6. On trichoscopy, short regrowing hair were more in the telogen effluvium (52.56%) than androgenetic alopecia (40.6%), however, this was not statistically significant (p=0.275).
7. The prevalence of peripilar sign on trichoscopy was found similar in androgenetic alopecia (18.10%) and telogen effluvium (17.63%).This was not statistically significant (p=0.962)
8. On trichoscan, the mean hair count was found to be lower in AGA (192.75 and 209 in the frontal and occipital scalp respectively) than in TE (200 and 212 in the frontal and occipital scalp respectively).

9. The median value of frontal vellus hair percentage was significantly higher in AGA (41.6) than in TE (21.95) and AGA+TE (28.2) (p=0.001).The median value of occipital vellus hair density was significantly higher in AGA (58.15) than in TE (32.1) (p=0.024).
10. The single follicular units were higher in AGA (49.44% in frontal scalp and 46.78% in occipital scalp) than in TE and AGA+TE group.
11. The median value of frontal hair thickness is lower in AGA (0.044 mm) than in TE (0.056 mm) and AGA+TE (0.051 mm), though this was not statistically significant. The median value of occipital hair thickness is significantly lower in AGA (0.042 mm) than in TE (0.055 mm) and AGA+TE (0.053 mm) (p=0.000).
12. The median value of frontal terminal: vellus ratio was significantly lower in AGA (1.4) than in AGA+TE (2.55) and TE (3.55) (p=0.001). Similarly median value of occipital terminal: vellus ratio was significantly lower in AGA (1.46) than in AGA+TE (2.63) and TE (3.85) (p=0.004).
13. The trichoscopic findings in alopecia areata showed numerous yellow dots in 63.9% patients, scanty yellow dots in 19.4% patients, black dots in 44.43%, broken hair in 63.9%, short vellus hair in 66.66% and short regrowing hair in 50% of patients.
14. The commonest trichoscopic findings in DLE were blue gray dots and globules (81%), loss of follicular units (84%) and structureless white areas (96%); and other common findings were hyperkeratotic plugs (69%) and telangiectasia (65%).

15. Yellow dots were highest in alopecia areata (83.33%) followed by androgenetic alopecia (56.4%) and then discoid lupus erythematosus (28%) followed in last by telogen effluvium (19.1%).
16. The majority (30.2%) of patients with hair loss fell into moderate to high disease activity based on SLEDAI.

LIMITATIONS

1. The patients included in our study were already on treatment for systemic lupus erythematosus; a prospective study including newly detected, treatment naive patients with SLE and follow up would have given us the exact magnitude of hair loss in this disease.
2. The histopathological examination was not done to confirm the diagnosis of the pattern of alopecia.
3. Hair clipping was not done in our patients before trichoscan which constrained us from calculating anagen verses telogen ratio.

RECOMMENDATIONS

1. Trichoscopy should be performed in all the SLE patients with alopecia.
2. Trichoscopy is helpful in categorising the diffuse non-scarring alopecia into different types.
3. Trichoscopy of DLE lesions of the scalp can be done to differentiate it from other types of scarring alopecia.

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ANNEXURES

ANNEXURE 1- A.R.A. CRITERIA FOR CLASSIFICATION OF S.L.E 1971

1. Facial erythema
2. Discoid lupus
3. Raynaud's phenomenon
4. Alopecia
5. Photosensivity
6. Oral or nasal ulceration
7. Arthritis without deformity
8. L.E. cells (2 or more)
9. Chronic false positive serological tests for syphilis (longer than 6/12)

10. Proteinuria (>3*5 g/day)
11. Cellular casts
12. Pleuritis or pericarditis
13. Psychosis or convulsions
14. Haemolytic anaemia or leucopenia (<4,000/mm) or thrombocytopenia (<100,000/mm)

ANNEXURE 2- THE 1982 REVISED CRITERIA FOR CLASSIFICATION OF SYSTEMIC LUPUS ERYTHEMATOSUS

Criterion	Definition
1. Malar rash	Fixed erythema, flat or raised, over the malar eminences, tending the spare the nasolabial folds
2. Discoid rash	Erythematous raised patches with adherent keratotic scaling and follicular plugging; atrophic scarring may occur in older lesions

3. Photosensitivity	Skin rash as a result of unusual reaction to sunlight, by patient history or physician observation
4. Oral ulcers	Oral or nasopharyngeal ulceration, usually painless, observed by a physician
5. Arthritis	Nonerosive arthritis involving two or more peripheral joints, characterized by tenderness, swelling, or effusion
6. Serositis	a. Pleuritis—convincing history of pleuritic pain or rub heard by a physician or evidence of pleural effusion Or b. Pericarditis—documented by electrocardiogram or rub or evidence of pericardial effusion
7. Renal disorder	a. Persistent proteinuria— >0.5 g/day or greater than 3+ if quantitation not performed Or b. Cellular casts—may be red cell, hemoglobin, granular, tubular, or mixed
8. Neurological disorder	a. Seizures—in the absence of offending drugs or known metabolic derangements (e.g., uremia, ketoacidosis, or electrolyte imbalance) Or

	b. Psychosis—in the absence of offending drugs or known metabolic derangements (e.g., uremia, ketoacidosis, or electrolyte imbalance)
9. Hematological disorder	a. Hemolytic anemia—with reticulocytosis Or b. Leukopenia— <4,000 μL total on two or more occasions Or c. Lymphopenia— <1,500/μL on two or more occasions Or d. Thrombocytopenia— <100,000 μL in the absence of offending drugs
10. Immunological disorder	a. Anti-DNA—antibody to native DNA in abnormal titer Or b. Anti-Sm—presence of antibody to Sm nuclear antigen Or c. Positive finding of antiphospholipid antibodies based on (1) an abnormal serum level of immunoglobulin G or immunoglobulin M anticardiolipin antibodies, (2) a positive test result for lupus anticoagulant using a standard method, or (3) a false-positive serologic test for syphilis known to be positive for at least 6 months and confirmed by Treponema pallidum immobilization or fluorescent treponemal antibody absorption test

11. Antinuclear antibody	An abnormal titer of antinuclear antibody by immunofluorescence of an equivalent assay at any point in time and in the absence of drugs known to be associated with “drug-induced lupus” syndrome

ANNEXURE 3- THE SLICC CRITERIA 2012

Clinical Criterion	Definition
1. Acute cutaneous lupus	Including: lupus malar rash (do not include if malar discoid), bullous lupus, toxic epidermal necrolysis variant of SLE, maculopapular rash, photosensitive lupus rash in the absence of dermatomyositis; or subacutue cutaneous lupus
2. Chronic cutaneous lupus	Including: classic discoid rash, hypertrophic (verrucous) lupus, lupus panniculitis (profundus), mucosal lupus, lupus erythematosus tumidus, chilblain lupus, discoid lupus/lichen planus overlap
3. Oral ulcers	Palate, buccal, tongue or nasal ulcers in the absence of other causes

4. Non-scarring alopecia	Diffuse thinning or hair fragility with broken hairs in the absence of other causes
5. Synovitis	Involving two or more joints characterized by effusion or swelling or tenderness in two or more joints and at least 30 min of morning stiffness
6. Serositis:	pleurisy or pericarditis More that 1-day duration of pleural/pericardial effusions or pleural/pericardial rub
7. Renal disorder:	Persistent proteinuria (>0.5 μg/day) or cellular casts
8. Neurological disorder	Seizures, psychosis, mononeuritis multiplex, myelitis or acute confusional state in the absence of other causes
9. Haemolytic anaemia	
10. Leukopenia or Lymphopenia	(<4000/ mm 3 at least once) or (<1000/mm3)

11. Thrombocytopenia	(<100 000/mm3 at least once)

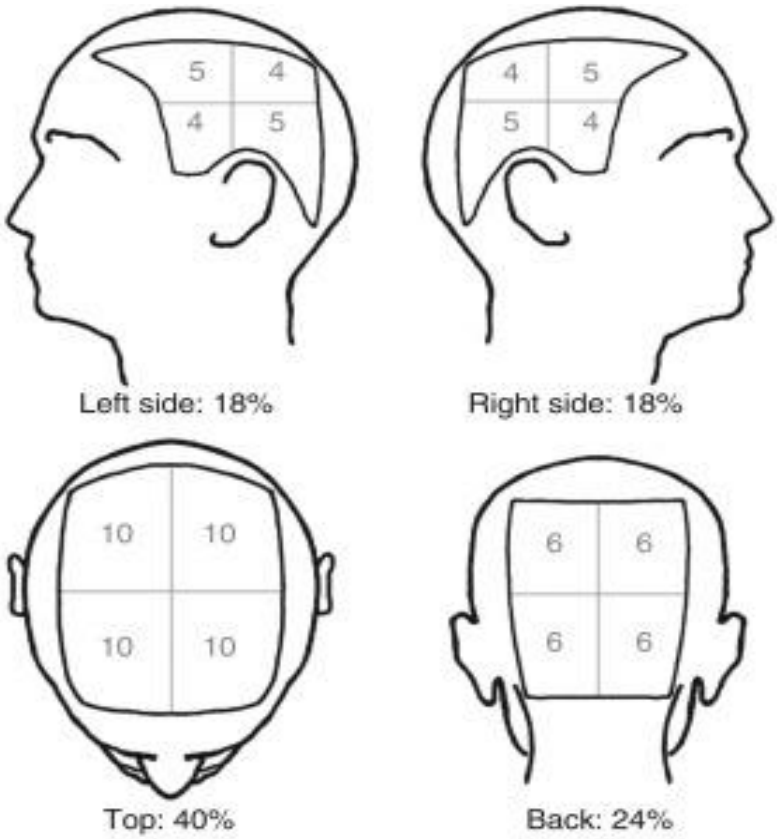
Immunological criteria
1. ANA above reference laboratory range
2. Anti-dsDNA antibody above reference laboratory range (or more than twofold the reference range if tested by ELISA)
3. Anti-Sm: presence of antibody to Sm nuclear antigen
4. Antiphospholipid antibody positivity
5. Low complement (low C3, C4 or CH50)
6. Direct Coombs' test in the absence of haemolytic anaemia

ANNEXURE 4- GILLIAM'S CLASSIFICATION

LE-Specific Skin Disease [Cutaneous LE (CLE)]	LE-Nonspecific Skin Disease
A. ACUTE CUTANEOUS LE (ACLE) 1. Localized ACLE (malar rash; butterfly rash) 2. Generalized ACLE (lupus maculopapular lupus rash, SLE rash, rash, photosensitive lupus dermatitis) **B. Subacute cutaneous LE (SCLE)** 1. Annular SCLE (syn. lupus marginatus, symmetric erythema centrifugum, autoimmune annular erythema, lupus erythematosus gyratus repens) 2. Papulosquamous SCLE (syn. disseminated DLE, subacute disseminated LE, superficial disseminated LE, psoriasiform LE, pityriasiform LE, and maculopapular photosensitive LE) **C. Chronic cutaneous LE (CCLE)** 1. Classic discoid LE (DLE) a. Localized DLE b. Generalized DLE 2. Hypertrophic/verrucous DLE 3. Lupus profundus/lupus panniculitis 4. Mucosal DLE a. Oral DLE b. Conjunctival DLE	**A. Cutaneous vascular disease** 1. Vasculitis a. Leukocytoclastic (1) Palpable purpura (2) Urticarial vasculitis b. Periarteritis nodosa-like cutaneous lesions 2. Vasculopathy a. Degos disease-like lesions b. Secondary atrophie blanche (syn. livedoid vasculitis, livedo vasculitis) 3. Periungual telangiectasia 4. Livedo reticularis 5. Thrombophlebitis 6. Raynaud phenomenon 7. Erythromelalgia (erythermalgia) **B. Nonscarring alopecia** 1. "Lupus hair" 2. Telogen effluvium 3. Alopecia areata **C. Sclerodactyly** **D. Rheumatoid nodules** **E. Calcinosis cutis** **F. LE-nonspecific bullous lesions** **G. Urticaria** **H. Papulonodular mucinosis** **I. Cutis laxa/anetoderma**

5. Lupus tumidus (urticarial plaque of LE) 6. Chilblain LE (chilblain lupus) 7. Lichenoid DLE (LE/lichen planus overlap, lupus planus)	**J. Acanthosis nigricans (type B insulin resistance)** **K. Erythema multiforme** **L. Leg ulcers** **M. Lichen planus**

ANNEXURE 5- SALT SCORE



Olsen/Canfield

Salt score			
Site:	Subject:	Visit:	Date:
Quadrant	Percentage involved	Multiplier	Score
Left side		0.18	
Right side		0.18	
Top		0.40	
Back		0.24	
Total			

ANNEXURE 6- SLEDAI SCORE

Appendix 1. SLEDAI-2K data collection sheet. (Check weight in SLEDAI-2K score column if descriptor is present at the time of the visit or in the preceding 10 days.)

Weight (check)	Descriptor	Definition
8 ❑	Seizure	Recent onset, exclude metabolic, infectious, or drug causes.
8 ❑	Psychosis	Altered ability to function in normal activity due to severe disturbance in the perception of reality. Include hallucinations, incoherence, marked loose associations, impoverished thought content, marked illogical thinking, bizarre, disorganized, or catatonic behavior. Exclude uremia and drug causes.
8 ❑	Organic brain syndrome	Altered mental function with impaired orientation, memory, or other intellectual function, with rapid onset and fluctuating clinical features, inability to sustain attention to environment, plus at least two of the following: perceptual disturbance, incoherent speech, insomnia or daytime drowsiness, or increased or decreased psychomotor activity. Exclude metabolic, infectious, or drug causes.
8 ❑	Visual disturbance	Retinal changes of SLE. Include cytoid bodies, retinal hemorrhages, serous exudates or hemorrhages in the choroids, or optic neuritis. Exclude hypertension, infection, or drug causes.
8 ❑	Cranial nerve disorder	New onset of sensory or motor neuropathy involving cranial nerves.
8 ❑	Lupus headache	Severe, persistent headache; may be migrainous but must be nonresponsive to narcotic analgesia.
8 ❑	Cerebrovascular accident	New onset of cerebrovascular accident(s); exclude arteriosclerosis.
8 ❑	Vasculitis	Ulceration, gangrene, tender finger nodules, periungual infarction, splinter hemorrhages, or biopsy or angiogram proof of vasculitis.
4 ❑	Arthritis	Two or more joints with pain and signs of inflammation (i.e., tenderness, swelling, or effusion).
4 ❑	Myositis	Proximal muscle aching/weakness, associated with elevated creatine phosphokinase/aldolase or electromyogram changes or a biopsy showing myositis.
4 ❑	Urinary casts	Heme granular or red blood cell casts.
4 ❑	Hematuria	More than five red blood cells/high power field; exclude stone, infection, or other cause.
4 ❑	Proteinuria	> 0.5 g/24 hr.
4 ❑	Pyuria	More than five white blood cells/high power field; exclude infection.
2 ❑	Rash	Inflammatory type rash.
2 ❑	Alopecia	Abnormal, patchy, or diffuse loss of hair.
2 ❑	Mucosal ulcers	Oral or nasal ulcerations.
2 ❑	Pleurisy	Pleuritic chest pain with pleural rub or effusion or pleural thickening.
2 ❑	Pericarditis	Pericardial pain with at least one of the following: rub, effusion, or electrocardiogram or echocardiogram confirmation.
2 ❑	Low complement	Decrease in the complement proteins C3 and C4 or in total complement activity (CH50), below the lower limit of normal for testing laboratory.
2 ❑	Increased DNA binding	Increased DNA binding by Farr assay above normal range for testing laboratory.
1 ❑	Fever	> 38°C; exclude infectious cause.
1 ❑	Thrombocytopenia	< 100,000 platelets/$\times 10^9$/L; exclude drug causes.
1 ❑	Leukopenia	< 3,000 white blood cells/$\times 10^9$/L; exclude drug causes.
	Total score	

Reproduced with permission from Gladman et al. (2002).

ANNEXURE 7- PROFORMA

Alopecia in Systemic Lupus Erythematosus Proforma

Date-

Sl.No/CHNo-

Informant-

History:

Name-

Age/Sex-

Address-

Marital status- unmarried/ married/ separated

History of present illness-

Duration of the disease- a) <1 year b) 1-2 year c) 2-3 years d) >4 years

Duration of the hair loss-

Approximate amount of hairloss per day- 0-50 / 50-100 / 100-200 / >200 per day

Hair loss– Episodic / Continuous/ Intermittent
Any trigger?
Yes/No
If yes – What was the trigger?.....

Did you notice your hair loss even before your disease was diagnosed?
Yes/No

Did you notice increase in hair loss with increase in your other symptoms of disease?
Yes/No

Was the hair loss patchy or diffuse?
Patchy/Diffuse

Is there any exacerbation of hair loss recently?
Yes/No

If yes.

Duration?.....Days/Months

Did you have scalp/skin lesions also with SLE?
Yes/No

Did your hair loss improve after initiating medications for SLE?
Yes/No

Do you have unwanted or excessive hair growth anywhere on your body?
Yes/No

Do you have thyroid problem?
Yes/No

If Yes.

Hypothyroidism/Hyperthyroidism

Does your scalp itch a lot or sometimes burn or hurt?
Yes/No

What do you think is the cause of your hair loss?

……
……..

Did you anytime have skin involvement?
Yes/No
If yes- localized / generalized

Did you have oral or nasal ulcers?
Yes/No

Do you have joint pains?
Yes/No

Do you have chest pain/ breathlessness/ abdominal pain?
Yes/No

Do you have renal involvement?
Yes/No

Past history-

Other comorbidities-Diabetes/ Hypertension/ Tuberculosis/ Bronchial asthma.

Any recent surgery in past 6 months?
Yes/No

Have you been under a severe amount of stress during the past 6 months?
Yes/No

Have you had any other serious illness during the past year?
Yes/No

If yes.

Diagnosis?.....

Personal history-

Have you started any special diets during the past year?
Yes/No

What are your food habits? Vegetarian/Non-vegetarian/Mixed diet

Do you get your menstrual period every month?
Yes/No

Any abortions.
Yes/ No
If yes – first/ second/ third trimester

How often do you wash/shampoo your hair? ……

Do your hair chemically processed or straightened?
Yes/No

Family history-

Do any of your family members also has SLE?
Yes/No

If yes.

Relation?.....

Do any of your family members also has hair loss?
Yes/No

Social history-

The disease affects your- daily work/ job/ studies/marriage/ nil

Did you changed your job/ stopped working/ stopped studying?
Yes/No

Treatment history-

Please tick the names of all the medications you are currently taking in the space below.
Steroids- Prednisolone/ Deflazacort/ Dexamethasone/ Hydrocortisone/ Methylprednisolone (Dose- ……)
Adjuvants- Mycofenolate mofetil/ HCQ/ Azathioprine (Dose-…….)
Others-

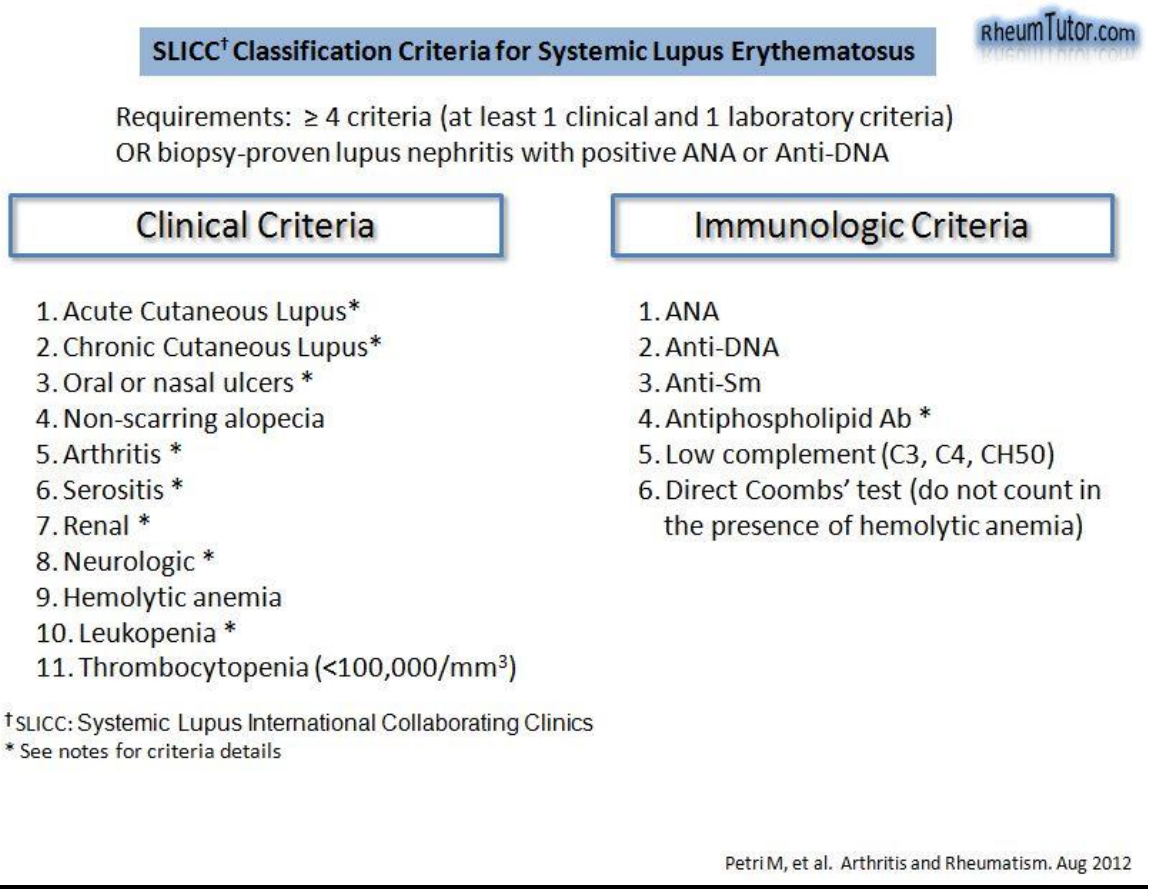
Please list any vitamins or natural products that you are taking:

……
…

Please list all the prescription and non-prescription treatments that you have tried for your hair loss condition:

……
……..

SLICC CRITERIA-



SLEDAI 2K-

Appendix 1. SLEDAI-2K data collection sheet. (Check weight in SLEDAI-2K score column if descriptor is present at the time of the visit or in the preceding 10 days.)

Weight (check)	Descriptor	Definition
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8 ❑	Cranial nerve disorder	New onset of sensory or motor neuropathy involving cranial nerves.
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8 ❑	Vasculitis	Ulceration, gangrene, tender finger nodules, periungual infarction, splinter hemorrhages, or biopsy or angiogram proof of vasculitis.
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4 ❑	Myositis	Proximal muscle aching/weakness, associated with elevated creatine phosphokinase/aldolase or electromyogram changes or a biopsy showing myositis.
4 ❑	Urinary casts	Heme granular or red blood cell casts.
4 ❑	Hematuria	More than five red blood cells/high power field; exclude stone, infection, or other cause.
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1 ❑	Leukopenia	< 3,000 white blood cells/$\times 10^9$/L; exclude drug causes.
	Total score	

Reproduced with permission from Gladman et al. (2002).

On examination-

General status –

BP-……mmHg

Pulse-……./min

Temperatue- Afebrile/ Febrile

Cutaneous examination-
Acute cutaneous LE – malar rash/ generalized rash
Subacute cutaneous LE- annular /papulosquamous lesions
Chronic cutaneous LE- Classic discoid LE – (Localized/ Generalized) / Hypertrophic /Lupus profundus /lupus panniculitis/ Mucosal DLE-
(Oral DLE/ conjunctival DLE) / Lupus tumidus/ Chilblain LE /Lichenoid DLE

Other cutaneous features-
Vasculitis /Vasculopathy/ Livedo reticularis/ Thrombophlebitis/ Raynaud phenomenon/ Erythromelalgia (erythermalgia)/
/Sclerodactyly/ Rheumatoid nodules/ Calcinosis cutis/ LE-nonspecific bullous lesions/ Urticaria/ Papulonodular mucinosis/ Cutis laxa/anetoderma/ Acanthosis nigricans/ Erythema multiforme/ Leg ulcers/ Lichen planus

Scalp- Non-scarring alopecia (diffuse/patchy/ lupus hair) / scarring alopecia

Mucosa- conjunctival congestion/ nasal ulcers/ oral ulcers

Nail- paronychia/ periungual telangiectasia

Joints- swelling/ erythema/ tenderness

Trichoscopic findings-

Black dots /Yellow dots/ Upright regrowing hairs/ Micro–exclamation mark hairs/ Vellus hairs/ Pigtail hairs (oval or circular)/ Broken hairs/ FollicularOpenings/ Monilethrix-like hairs/ Trichorrhexis nodosa

Hair thickness heterogeneity/ Thin hairs/ Vellus hairs/ Single-hair pilosebaceous units/ Yellow dots/Perifollicular discoloration/ Wavy hair/Honeycomb pigmentation

Predominance of follicular units with only one hair/ Perifollicular discoloration (peripilar sign)/ Empty hair follicles/yellow dots/ Lack of features typical of other diseases

Thick arborizing vessels/ Large yellow dots (follicular keratotic plugs)/ Fine interfollicular scaling/ Scattered brown discoloration/ Red dots/ Blue-gray dots (on dark or sun-exposed skin)/ Loss of follicular openings/ Pink areas/ White areas/ Arborizing vessels/ Yellow dots containing thin spider vessels

Investigations-

TSH
CBC
IRON
FERRITIN
VITAMIN B12
VITAMIN D

Final diagnosis-

ANNEXURE 8- PATIENT INFORMATION SHEET

Study Title: Clinical and trichoscopic patterns of alopecia in systemic lupus erythematosus.

Study title (for lay public): Study of different types of hair loss seen in systemic lupus erythematosus and their features under magnification.

We are inviting you to take part in a research study. Before you decide it is important for you to understand why we are doing the research and what it will involve. Please read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or if you want more information. Take time to decide whether you wish to take part or not.

Purpose of research:

We are going to do a study on different types of hair loss in systemic lupus erythematosus. There are different patterns of hair loss in SLE which can be temporary or permanent. Few studies have shown that temporary hair loss in SLE can be associated with increased disease severity. There may be certain features of the hair and scalp which can be associated with disease severity. We want to do research on this topic because there is little information on this in India. This study will provide data on this topic and helps for better understanding of not only different types of hair loss in SLE but also it's correlation with the disease severity. This will be useful for optimum management and early recognition of such conditions.

Expected duration of the Subject's participation:

You will be examined by the doctor only once in the study period.

Description of the procedures:

The doctor will do the detailed physical examination and will note down the information in a special form.

Relevant blood investigations which includes complete blood profile, iron, ferritin, vitamin B12, vitamin D, thyroid function tests will be done depending on the clinical diagnosis. Examination of the hair and scalp in different magnifications will be done and photographs will be taken using a special instrument called Fotofinder. The SLE disease severity will be assessed using a scoring system. At the end of the study, we will analyze all the data obtained so far and correlate with the disease severity.

Risks or discomforts to the Subject:

Small amount of blood will be collected from peripheral vein. Possible complications include

- Pain
- Bleeding at the puncture site
- There is also a slight possibility of blood clot under skin.

We are not doing any additional tests apart from the standard protocol.

Benefits to the Subject:

We are going to study the hair loss types and do tests like Complete blood profile, Iron, Ferritin, Vitamin B12, Vitamin D, Thyroid function tests. In this study we are going to know the type of hair loss and its microscopic patterns which will guide us to the disease severity and will be helpful in making a decision regarding treatment.

Benefits to others:

Overall data about hair loss patterns in SLE is very little in India. This study will provide data on this topic and helps for better understanding of different types of hair loss seen in SLE. This will be useful for early recognition and optimum management of such condition.

Confidentiality:

Your name and address will not be revealed at any point of time. All information which is

collected about you during the course of the research will be kept strictly confidential unless we

are required by law to share any information.

Participation:

It is up to you to decide whether to take part or not. You are still free to withdraw from the

study at any time, without giving any reason. A decision to withdraw, or a decision not to take

part, will not affect the standard of care you receive.

Contact person:

Dr. Shivani Bhardwaj

Post Graduate resident,

Department of Dermatology Venereology and Leprosy,

Christian Medical College and Hospital,

Vellore.

Mobile- 9677607462

Office-04162282054

ANNEXURE 9- INFORMANT CONSENT FORM

Informed Consent form to participate in a clinical trial

Study Title: Clinical and trichoscopic patterns of alopecia in systemic lupus erythematosus

Subject's Initials: _____ Subject's Name: _____

Date of Birth / Age: _____

(i) I confirm that I have read and understood the information sheet dated _____ for the above study and have had the opportunity to ask questions. [ ]

(ii) I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. [ ]

(iii) I understand that the Ethics Committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further

research that may be conducted in relation to it, even if I withdraw from the trial. I agree to this

access. However, I understand that my identity will not be revealed in any information released

to third parties or published. [ ]

(iv) I agree not to restrict the use of any data or results that arise from this study provided such

a use is only for scientific purpose(s) [ ]

(v) I agree to take part in the above study. [ ]

Signature (or Thumb impression of the Subject/Legally Acceptable Representative):

Signatory's Name: _____ Date: ____/____/_____

Signature of the Investigator: _____

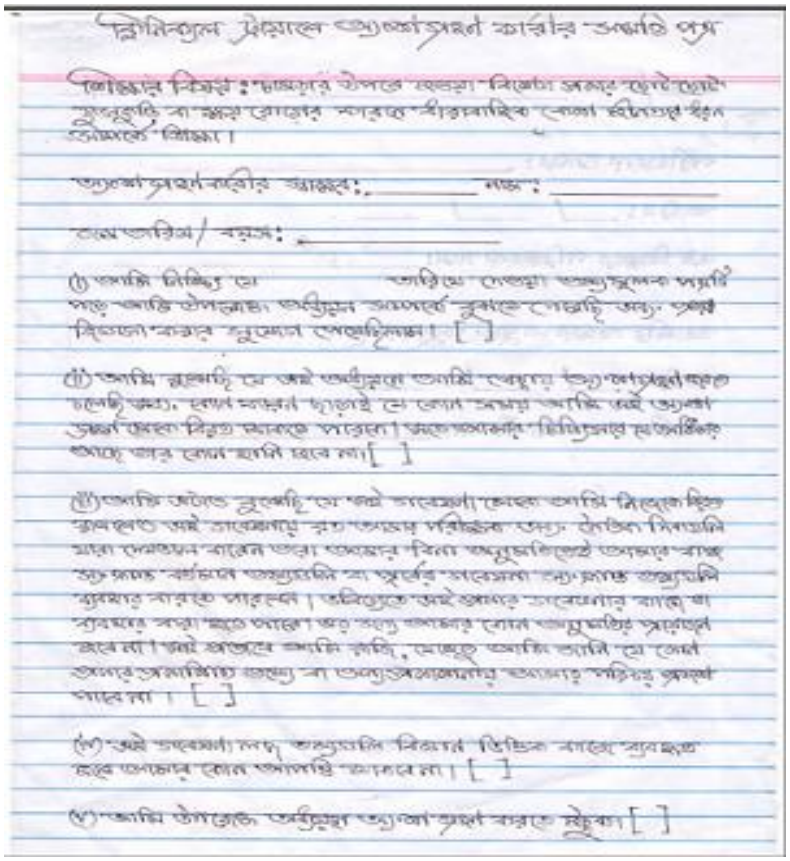
Date: _____/_____/_____

Study Investigator's Name: _____

Signature or thumb impression of the Witness: _____

Date: _____/_____/_____

Name & Address of the Witness: _____



গবেষণায় অংশগ্রহণ কারীর সম্মতি পত্র

শিরোনাম বিষয়ঃ ..... নম্বর: .....

তারিখ/ বয়স: .....

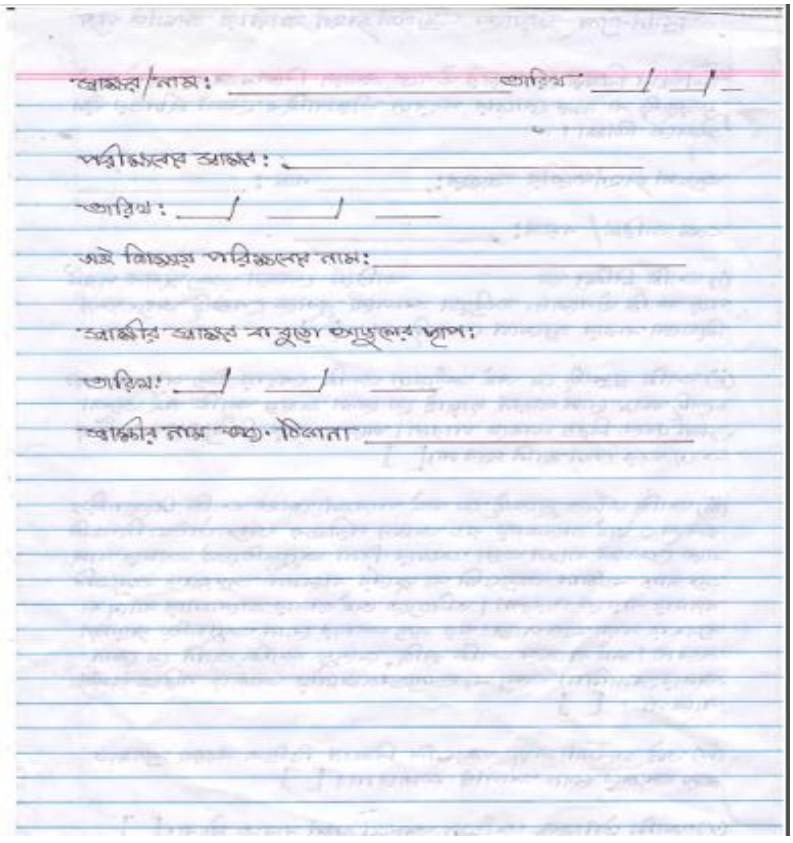
(i) আমি নিশ্চিত যে ..... সম্পর্কে বুঝতে পেরেছি এবং প্রশ্ন করার সুযোগ পেয়েছিলাম। [ ]

(ii) আমি বুঝেছি যে আমি স্বেচ্ছায় এই গবেষণায় অংশগ্রহণ করতে চলেছি এবং যে কোন সময় আমি এই গবেষণা থেকে বিনা কারণে প্রত্যাহার করতে পারব। [ ]

(iii) আমি এটাও বুঝেছি যে এই গবেষণা থেকে আমি নিজেকে বিচ্ছিন্ন করলেও এই গবেষণায় প্রাপ্ত তথ্যের পরিচ্ছন্নতা রাখা হবে। [ ]

(iv) এই গবেষণা সম্পর্কিত কাগজপত্রে আমার নাম ব্যবহৃত হলে আমার কোন আপত্তি থাকবে না। [ ]

(v) আমি স্বেচ্ছায় এই গবেষণায় অংশগ্রহণ করতে সম্মত। [ ]



স্বাক্ষর/নাম: ..... তারিখ: ___/___/___

পরীক্ষকের স্বাক্ষর: .....

তারিখ: ___/___/___

এই শিশুর পরিচ্ছদের নাম: .....

সাক্ষীর স্বাক্ষর বা বুড়া আঙ্গুলের ছাপ:

তারিখ: ___/___/___

সাক্ষীর নাম ও ঠিকানা: .....

चिकित्सकीय परीक्षण में भाग लेने के लिए सूचित सहमति पत्र

अध्ययन शीर्षक: एस.एल.ई. में असमय बाल झड़ने का नैदानिक तथा ट्राईकोस्कोपिक पेटर्न का अध्ययन

प्रतिभागी का नाम: _____
जन्मतिथि / उम्र: _____

१) मैं इस बात की पुष्टि करता हूं कि मैंने दिनांक _____ सूचना पत्र को उपरोक्त अध्ययन के लिए पढ़ा और समझा है, और मुझे प्रश्न पूछने का मौका मिला है।

२) मैं इस बात को समझता हूं कि इस अध्ययन में मेरी भागीदारी स्वैच्छिक है। मैं किसी भी समय बगैर कोई कारण बताए, तथा मेरी चिकित्सा में बिना कोई बाधा आए, या कानूनी अधिकार बिना प्रभावित हुए, इस अध्ययन को छोड़ सकता हूं।

३) मैं समझता हूं कि आचार समिति और नियामक अधिकारियों को मेरे स्वास्थ्य अभिलेखों के वर्तमान अध्ययन और इस संबंध में भविष्य में होने वाले अनुसंधानों के लिए मेरी अनुमति की जरूरत नहीं होगी, चाहे मैं इस से अपनी भागीदारी वापस ले लूं। मैं इस बात से सहमत हूं।

हालांकि, मैं समझता हूं मेरी पहचान का खुलासा तीसरे पक्ष को दी गई किसी भी जानकारी अथवा प्रकाशन में नहीं किया जायगा

४) मैं इस बात के लिए सहमति देता हूं कि इस अध्ययन से उत्पन्न हुए परिणामों को वैज्ञानिक प्रायोजन के लिए प्रदान करने से इंकार नहीं करूंगा।

५) मैं उपरोक्त अध्ययन में भाग लेने के लिए सहमत हूं।

हस्ताक्षर (विषय / कानूनी तौर पर स्वीकार्य प्रतिनिधि का या अंगूठे का निशान):

हस्ताक्षरकर्ता का नाम: _____ दिनांक: ___ / ___ / ____

अन्वेषक के हस्ताक्षर: _____

तारीख: ___/___/____

अध्ययन जांचकर्ता का नाम: _____

गवाह का हस्ताक्षर या अंगूठे का निशान: _____

तारीख: ___/___/____

नाम व गवाह का पता: _____

ഗവേഷണ പഠനത്തിൽ പങ്കെടുക്കുന്നതിനുള്ള

പഠനം :- ഈ ഗോത്രത്തിൽ നടത്തുന്ന പഠനത്തിനുള്ള
വിവരശേഖരണത്തിന്

വ്യക്തിയുടെ പേര് :-

ജനന തീയതി / വയസ്സ് :-

(i) ഞാൻ ഈ പഠനത്തിനായി ചോദിക്കുന്ന കാര്യങ്ങൾ
_ _ _ _ എന്നെ തിരിച്ചറിയുന്ന പ്രത്യേക രീതിയിൽ
മനസ്സിലാക്കിയെന്നും ഇതിനെക്കുറിച്ചുള്ള എന്റെ
സംശയങ്ങൾ ചോദിക്കാനുള്ള അവസരം എനിക്ക്
ലഭിച്ചുവെന്നും മനസ്സിലാക്കുന്നു.

(ii) ഈ പഠനത്തിലുള്ള എന്റെ പങ്കാളിത്തം തീർത്തും
എന്റെ തീരുമാനമാണെന്നും എന്നു പിൻവലിയും
എനിക്ക് ഇതിൽ നിന്നും ഒരു കാരണവും വെളിപ്പെടുത്താതെ
- തന്നെ പിൻമാറാനുള്ള സ്വാതന്ത്ര്യം ഉണ്ടെന്നും
മനസ്സിലാക്കുന്നു. പിൻവാങ്ങുമ്പോൾ എനിക്ക നൽകുന്ന
ചികിത്സയിലും മറ്റ് ഒരു സൗകര്യങ്ങളും മാറ്റമില്ലാതെ തുടരുമെന്നും
ഞാൻ മനസ്സിലാക്കുന്നു.

(iii) എന്റെ ഫലങ്ങൾ മറ്റാരുമായുള്ള പ്രസിദ്ധീകരണ
- രേഖ അനുവാദം കൂടാതെ ഈ പഠനത്തിനുള്ള
സഹപ്രവർത്തകർക്കും എന്നിലൂടെ മറ്റിടി അധികൃതർക്കും
ആവശ്യമില്ലെന്നും ഞാൻ മനസ്സിലാക്കുന്നു.
ഈ പഠനത്തിൽ നിന്നും പിൻവാങ്ങിയാലും
അതിനുമുമ്പ് അവരുടെ അനുവാദം അവർക്കുണ്ടെന്നും
ഇതിനെക്കുറിച്ചു സംബന്ധമുള്ള പുതിയ പഠനങ്ങളിലും
എന്നെ എന്റെ റെഗുലർവൈസ് അവർക്ക്
പരിശോധിക്കാനും കഴിയും. ഞാൻ ഇനിയും
സമയം നൽകുന്നു. എന്നാൽ എന്റെ
പേരോ മറ്റു വിശദാംശങ്ങളൊ വെളിപ്പെടുത്തുന്നില്ല

(iv) ഗവേഷണ ഉദ്ദേശ്യത്തിനായി ഉപയോഗിക്കാൻ വേണ്ടി
രക്ത ഫലവും മറ്റുള്ളവരുടെ രക്തവുമുൾപ്പെ എന്ന്
ഞാൻ സമ്മതിക്കുന്നു.

(v) ഞാൻ ഈ പഠനത്തിൽ പങ്കെടുക്കാൻ
സമ്മതിക്കുന്നു.

ഒപ്പ് (പങ്കെടുക്കുന്നയാളുടെ ബന്ധു ചേർക്കുക)

പേര് :- തീയതി :-

പഠനം നടത്തുന്ന ആളുടെ പേര് :-

[redacted]

(ii)

ഒപ്പ് (രക്ഷാകർത്തി)

തീയതി.

രക്ഷാകർത്തിയുടെ
പേരും വിലാസവും :-

மருத்துவ ஆராய்ச்சியில் பங்கேற்பதற்கான ஒப்புதல் படிவம்

ஆய்வின் தலைப்பு :
சிஸ்டமிக் ஸ்டீபல் எரிதமாடோசஸ்னால் ஏற்படும் வழுக்கை மற்றும் Trichoscopic வடிவங்களை பற்றிய மருத்துவ ஆய்வு.

பங்கேற்பாளரின் எண்: _____

பங்கேற்பாளரின் பெயர் :_____

பிறந்த தேதி / வயது :_____/_____

1. இந்த ஆராய்ச்சியில் நான் _____ தேதியில் மேற்கண்ட தகவல்படிவத்திலுள்ள அனைத்துதகவல்களையும் நன்கு படித்து புரிந்துகொண்டேன். மேலும் கேள்விகள் கேட்க வாய்ப்ளிக்கப்பட்டது.

2. இந்த ஆராய்ச்சியில் பங்குகொள்வது என் விருப்பம்சார்ந்தது என்பதனையும், இவ்வாராய்ச்சியில் இருந்து எப்போது வேண்டுமானாலும் எக்காரணமும் இன்றி விலக்கிகொள்ளலாம் என்பதையும் புரிந்துகொண்டேன். எனுடைய விலகல் என் மருத்துவ சிகிச்சைக்கான எந்த ஒரு உரிமையையும் பாதிக்காது என்பதையும் புரிந்துகொண்டேன்.

3. இந்த ஆராய்ச்சி சம்பந்தமான தொடர்பில் உள்ளவர்கள் சட்டபூர்வமான குழுவைச்சார்ந்தவர்கள் மற்றும் ஒழுங்குமுறை குழுவைசார்ந்தவர்கள் என்பதையும் எனுடைய மருத்துவ பதிவேடுகளை என் அனுமதியின்றி பயன்படுத்தலாம் என்பதற்கு முழு சம்மதத்தைதொரிவித்துகொள்கிறேன். நான் இந்த ஆய்வில் இருந்து விலகினாலும் பதிவேடுகளை பயன்படுத்தலாம். மேலும் ஆய்வின் முடிவுகளை வெளியிடும் பொழுது எனைன குறித்த தகவல்கள் வெளியிடப்படாது என்பதையும் புரிந்துகொண்டேன்.

4. இந்த ஆராய்ச்சியில் பெறப்படும் தகவல்கள் மற்றும் முடிவுகள அறிவியல் சம்மந்தமாக பயன்படுத்துவதில் எனக்கு எந்த மறுப்பும் இல்லை.

இரகசியத்தன்மை :
இந்த ஆய்வில் உங்கள் பெயரோ அல்லது முகவரியோ எந்த இடத்திலும் குறிப்பிடப்படாது. இந்த ஆய்வின் பொழுது உங்களைப்பற்றி பெறப்பட்ட அனைத்து தகவலும் மிகவும் பாதுகாப்பாகவும், இரகசியமாகவும் வைக்கப்படும். மேலும் இத்தகவலை தேவைப்படும் பொழுது சட்டவிதிமுறைக்குட்பட்டே பயன்படுத்தபடும்.

பங்குபெறுவது :
இந்த ஆய்வில் நீங்கள் பங்குகொள்ளுதல் அல்லது பங்குபெறாமுத்தல் உங்களுடைய சுயவிருப்பமே. இந்த ஆய்வின் பொழுது எக்காரணமும் இன்றி, எந்த நேரத்திலும் நீங்கள் தானாகவே விலகிக்கொள்ளலாம். மேலும் நீங்கள் விலகினாலோ அல்லது பங்குபெற மறுத்தாலோ அதனால் தாங்கள் பெறும் மருத்துவ சிகிச்சைய முறையில் எந்த விதமான பாதிப்பும் ஏற்படாது.

அணுகவும்:

டாக்டர். **வினாஸி பரத்வாஜ்**
தோல்நோய் துறை,
கிருத்துவ மருத்துவ கல்லூரி, வேலூர்.
தொலைபேசி எண்: 0416 2282054
கைபேசி எண்: 9677607462.

5. நான் இந்த ஆராய்ச்சியில் பங்குகொள்ள முழு மனதுடன் சம்மதிக்கிறேன்.

நோயாளியின் கையொப்பம்/ கைரேகை:.....

தேதி:...../...../.....

நோயாளியின் பெயர்: .....

சாட்சியாளரின் கையொப்பம்/ கைரேகை:..... தேதி:...../...../.....

சாட்சியாளரின் பெயர் மற்றும்

முகவரி:_____

_____

ஆய்வாளரின் கையொப్పம்/ கைரேகை:.....

தேதి:...../...../.....

ఆய్వాளரின் பெயர்: .....

అంగీకార పత్రం

అధ్యయన శీర్షిక: SLE లో ఎముక కణం జబ్బు రాని సమస్యలు మరియు వాటి ట్రైగ్నాస్టిక్ లక్షణాల అధ్యయనం.

- అధ్యయన సంఖ్య:
- వ్యక్తి ఇంటి పేరు:
- వ్యక్తి పేరు:
- పుట్టిన తేదీ/ వయస్సు:

1) _____ తేదీ నాటి ఈ అధ్యయనానికి సంబంధించిన సమాచార పత్రము చదివి, అర్థం చేసుకొని, ప్రశ్నలడిగే అవకాశం దొరికి సంతృప్తిపొందాను.

2) ఈ అధ్యయనంలో పాల్గొనడం పూర్తిగా వ్యక్తిగతం అని, ఏ సమయంలోనైనా కారణం చెప్పకుండా వైదొలగొచ్చని, పరిశోధనా కాలం సంబంధిత బాధ్యతలు మీద ఏ ప్రభావం లేకుండా నేను అర్థం చేసుకున్నాను.

3) ఈ అధ్యయనంలో నేను ఇచ్చే వివరాలు, X-కిరణాల రూపాలు మరియు రిపోర్టులు ఎప్పుడూ నా వ్యక్తి సంబంధిత సమాచారాన్ని నా అనుమతి లేకుండానే చూడవచ్చు. అందుకు నేను అంగీకరిస్తున్నాను. నా వ్యక్తిగత సమాచార గోప్యత కూడా ప్రమాదంలో పడకుండా నేను అర్థం చేసుకున్నాను.

4) ఈ అధ్యయనానికి సంబంధించిన సమాచారం లేదా ఫలితాలు, శాస్త్రీయ ప్రయోజనాల కొరకు ఎప్పుడంటే ప్రచురితులు లేకుండా ఉపయోగించుటకు అంగీకరిస్తున్నాను.

5) ఈ అధ్యయనంలో పాల్గొనుటకు అంగీకరిస్తున్నాను

- వ్యక్తి సంతకం (లేదా వేలిముద్ర):
  వ్యక్తి పేరు: తేదీ:
- వైద్యుని సంతకం:
  వైద్యుని పేరు: తేదీ:
- సాక్షి సంతకం (లేదా వేలి ముద్ర):
  సాక్షి పేరు: తేదీ:

ANNEXURE 10- ABSTRACT

INTRODUCTION

Alopecia is one of the commonest finding in Systemic Lupus Erythematosus (SLE) and this constitutes one of the diagnostic clinical criterion in Systemic Lupus International Collaborating Clinic (SLICC) criteria.

OBJECTIVE

To observe the clinical and trichoscopic patterns of different types of alopecia seen in Systemic Lupus Erythematosus.

To study the patterns of hair loss in systemic lupus erythematosus and its correlation with SLE disease activity index (SLEDAI score).

METHODS

Ninety six patients of SLE who had alopecia were enrolled during the study period between September 2016 and August 2017. The clinical pattern of alopecia as diffuse non-scarring alopecia, alopecia areata and discoid lupus erythematosus were noted. Trichoscopy was done for all the patients on the frontal and occipital scalp. The patients with hair diameter diversity (HDD) more than 20% were classified as androgenetic alopecia (AGA). The patients who did not have HDD more than 20% but had diffuse alopecia were classified as telogen effluvium (TE). The other features looked for were vellus hair, predominant single follicular units, yellow dots, peripilar sign and short regrowing hair. Trichoscan was conducted in diffuse non-scarring alopecia to correlate

the findings. Trichoscopy was done from all the patches of alopecia areata and from the most representative site of discoid lupus erythematosus. SLEDAI scoring was calculated for all the patients.

RESULTS

There were 96 patients of SLE with alopecia. The majority of patients with diffuse non-scarring alopecia had onset of hair loss in third decade of life i.e. between 21-30 years. The female to male ratio was 6:1. There were 14 patients (17.94%) who had clinically evident AGA and 14 patients (17.94%) with lupus hair. There were 19 patients with AGA, 46 patients with TE and 13 patients with the combination of AGA and TE. There were 6 patients with alopecia areata and 26 patients with discoid lupus erythematosus.

CONCLUSION

The most common cause of alopecia in our SLE patients was chronic telogen effluvium. Trichoscopy is an effective tool in diagnosing different patterns of alopecia in SLE.

ANNEXURE 11- DATA SHEET

slno	date	omchno	infrmt	name	age	ag	sex	marstat	indian	state	durxn	ao	durallop	aoa	amontalo	corsallop	trig	trigger	alopbdx	alopv
1	########	332776G	1	HONEY AN	22	2	1	2	1	8	2	20	2	20	3	1	2	NIL	2	
2	########	795498G	1	SAPNA MI	32	2	1	1	1	2	9	23	2	30	3	1	2	NIL	2	
3	########	200879G	1	SILUVAI M	45	3	1	1	1	1	1	44	3	42	4	2	2	NIL	2	
4	########	861514G	1	SUDHA K	32	2	1	1	1	1	0.03	32	0.03	32	3	2	2	NIL	2	
5	########	675396C	1	BHARATHI	36	2	1	1	1	1	10	26	8	28	4	2	2	NIL	1	
6	########	520383G	1	SWATI LEI	30	2	1	1	1	2	1	29	1	29	4	2	2	NIL	2	
7	########	170667g	1	KANAGA S	28	2	1	1	1	1	2	26	1	27	3	2	2	NIL	2	
8	########	350225G	1	NITESH KU	18	1	2	2	1	5	3	15	0	18	1	4	2	NIL	2	
9	########	503735C	1	SELVARAN	52	4	1	1	1	1	10	42	10	42	4	2	2	NIL	2	
10	########	859166G	1	KOHINOO	40	3	1	1	1	2	8	32	8	32	2	2	2	NIL	2	
11	########	740768G	1	SURJI DEV	32	2	1	1	1	6	1	31	1	31	4	2	2	NIL	2	
12	########	224988F	1	SASWATI I	28	2	1	2	1	7	5	23	4	24	3	3	2	NOT NOTI	2	
14	########	915686G	1	PAPIA DAS	29	2	1	1	1	2	0.06	28	0.03	29	2	2	2	NA	2	
15	########	035799F	1	SARAYU S	21	2	1	2	1	1	6	15	2	19	4	2	2	NA	2	
16	########	710282G	1	ANANYA D	40	3	1	1	1	2	0.05	40	0.05	40	4	2	2	NA	2	
17	########	669598G	1	ANJU SING	28	2	1	1	1	1	1	27	0.02	28	4	1	2	NA	2	
18	########	331522f	1	ANNAPOO	29	2	1	1	1	3	4	25	4	25	3	2	2	NA	2	
19	########	115676C	1	ASHA PRA	38	2	1	1	1	2	15	23	0.06	38	2	2	2	NA	2	
20	########	754991G	1	ALISHA	23	2	1	1	2	9	3	20	3	20	3	2	2	NA	2	
21	########	894276G	1	ASMA KHA	31	2	1	1	1	6	5	26	4	27	4	1	1	FEVER	2	
22	########	056413G	1	ASTMA DE	22	2	1	1	1	6	2	20	2	20	4	2	2	NIL	2	
23	########	862014F	1	ARUN	26	2	2	2	1	1	3	23	0	26	1	4	2	NO HAIRL	2	
23	########	862014F	1	ARUN	26	2	2	2	1	1	3	23	0	26	1	4	2	NO HAIRL	2	
24	########	686508D	1	BASREEN	34	2	1	1	1	1	7	27	0.02	34	2	2	2	NIL	1	
25	########	648634G	1	BHAKTI PA	25	2	1	1	1	2	2	23	0.06	25	2	3	1	FEVER	2	
26	########	309761F	1	BHUVANE	45	3	1	1	1	1	4	41	0.06	45	4	3	2	NIL	2	
27	########	124570D	1	BIPAD BAI	46	3	2	1	1	7	9	37	10	36	4	3	2	NIL	1	
28	########	497742G	1	BINAKSHI	54	4	1	1	1	7	0.01	54	0.01	54	3	2	2	NIL	2	
29	########	318100F	1	CLARA	51	4	1	1	1	1	3	48	2	49	2	2	2	NIL	2	
30	########	262488c	1	CHANDAN	24	2	1	1	1	2	12	12	2	22	2	1	2	NIL	1	
32	########	524161G	1	DEVAKI DE	23	2	1	1	1	3	1	22	1	22	4	2	2	NIL	2	
33	########	455440F	1	EVANGELI	26	2	1	2	1	1	3	23	3	23	3	3	2	NIL	2	
34	########	836243g	1	GITA DAS	56	4	1	1	1	2	12	44	10	46	4	1	2	NIL	2	
35	########	323990G	1	GURVINDE	30	2	1	1	1	2	3	27	1	29	4	2	1	WITH SKIN	2	
36	########	530549G	1	JASMINA I	28	2	1	1	1	2	3	25	3	25	4	2	2	NIL	2	
37	########	867166G	1	JASMINE I	35	2	1	1	1	1	1	34	0.04	35	3	3	2	NA	2	
38	########	928407F	1	JANS BAN	32	2	1	1	1	1	2	30	2	30	4	1	2	NIL	2	
39	########	518919G	1	JAYAPRIYA	27	2	1	1	1	1	0.09	26	0.09	27	3	3	2	NIL	2	
40	########	887802G	1	JEBRANI	18	1	1	2	1	1	0.01	18	0.04	18	4	2	2	NA	2	
41	########	703687d	1	KANCHAN	52	4	1	1	1	1	7	45	7	45	4	2	2	NA	2	
42	########	114328G	1	KUNCHOK	38	2	1	1	1	7	2	36	2	36	2	3	2	NA	2	
43	########	872270D	1	LALLIANPI	46	3	1	1	1	7	4	42	4	42	3	2	2	NA	2	
44	########	880057G	1	LALNUNPA	26	2	1	2	1	7	1	25	0.05	26	2	2	2	NA	2	
45	########	678071G	1	LINDA LAL	29	2	1	2	1	7	1	28	0.02	29	3	2	1	JOINT PAI	1	
46	########	808167D	1	MARIAMM	40	3	1	1	1	1	11	29	11	29	4	2	2	NIL	1	
46	########	808167D	1	MARIAMM	40	3	1	1	1	1	11	29	11	29	4	2	2	NIL	1	
47	########	478730D	1	MANASI M	28	2	1	1	1	2	4	24	4	24	3	1	1	WITH SLE	2	
48	########	651809D	1	MADOA CI	40	3	2	1	1	3	6	34	6	34	3	1	1	JAUNDICE	1	
49	########	052176B	1	MADHUBA	24	2	1	2	1	1	9	15	9	15	3	2	2	NIL	2	
50	########	029256G	1	MANIMAL	20	2	1	2	1	2	4	16	4	16	2	1	1	WITH SKI I	2	
51	########	625860G	1	MD ABRAI	18	1	2	2	2	9	1	17	0.04	18	2	2	2	NIL	1	
53	########	898708G	1	MISHA MI	26	2	1	2	1	1	0.08	25	0.08	26	4	1	2	NIL	2	
54	########	274402G	1	MOUMITA	23	2	1	2	1	2	1	22	8	15	2	2	2	NIL	1	
55	########	636380G	1	MAYNA RI	29	2	1	1	1	2	1	28	1	28	1	2	2	NIL	2	
56	########	957443D	1	NAJMABI	30	2	1	1	1	1	7	23	6	24	3	3	2	NIL	2	
57	########	808158g	1	NAGAMAI	43	3	1	1	1	3	12	31	12	31	4	2	2	NIL	2	
58	########	406401C	1	NIRMALA	38	2	1	2	1	1	17	21	17	21	3	2	2	NIL	2	
59	########	619211C	1	NIRMALA	30	2	1	1	1	1	11	19	11	19	3	3	2	NIL	2	
60	########	995760F	1	NANDHIN	24	2	1	2	1	1	2	22	2	22	3	2	2	NIL	2	
61	########	562446G	1	MANJULA	40	3	1	1	1	1	3	37	3	37	3	2	2	NIL	2	
62	########	857808G	1	MST SHAH	38	2	1	1	2	9	1	37	0.06	38	4	2	2	NIL	2	
63	########	861977G	1	PRAKASH	42	3	2	1	1	2	0.02	42	0	42	1	4	2	NIL	2	
64	########	844175G	1	PRATIMA	44	3	1	1	1	2	3	41	1	43	1	1	1	MEDICINE	2	
65	########	600935C	2	PARKAVI I	19	1	1	2	1	1	12	7	2	17	3	1	2	NIL	2	
66	########	559512G	1	PREMALA	34	2	1	1	1	1	1	33	0.01	34	4	2	2	NIL	2	
67	########	618680F	1	RADHIKA.	31	2	1	1	1	1	4	27	0.03	31	3	1	2	NIL	2	
68	########	452051F	1	RAJASHRE	26	2	1	2	1	7	3	23	3	23	2	2	2	NIL	2	
69	########	704476G	1	RAVI	33	2	2	1	1	7	2	31	9	24	1	2	2	NIL	1	

70	########	111182G	1	REUBEN C	34	2	2	1	1	1	8	26	8	26	2	1	2	NIL	1	
71	########	276162f	1	RINA DEVI	30	2	1	1	1	5	5	25	6	24	4	2	2	NIL	2	
72	########	720011G	1	ROHINI KH	18	1	1	2	1	2	1	17	0.06	18	1	2	2	NIL	2	
73	########	879238G	1	ROJA	37	2	1	1	1	1	2	35	0	37	1	4	2	NIL	2	
74	########	882609G	1	RUMPA SI	34	2	1	1	2	9	0.06	34	0.06	34	1	2	2	NIL	2	
76	########	385632F	1	SWAPANA	46	3	1	1	1	6	4	42	6	40	2	1	2	NIL	2	
77	########	613380D	1	SANTHOSI	26	2	2	2	1	1	8	18	5	21	2	2	2	NIL	2	
78	########	7246948	1	SARBANI (	36	2	1	1	1	5	18	18	17	19	4	3	1	RAINY SEA	1	
79	########	171547C	1	SUPRAVA	33	2	1	1	1	7	5	28	5	28	4	1	1	SLE SYMPT	2	
80	########	812750G	1	SIVAKUMA	22	2	1	1	1	3	1	21	0.02	22	3	2	2	NIL	2	
81	########	800461G	1	SHAILYA K	34	2	1	2	1	8	0.08	33	1	33	3	2	2	NIL	2	
82	########	273899D	1	SHIPRA RA	49	3	1	1	1	2	0.06	49	1	48	3	3	1	FEVER	1	
83	########	805027C	1	SHRABAN	27	2	1	1	1	2	11	16	3	25	3	2	2	NIL	2	
85	########	303827C	1	SONALI DI	30	2	1	1	1	2	13	17	1	29	1	1	1	WITH SLE	2	
86	########	732930G	1	SUNAINA	27	2	1	2	1	1	12	15	0.08	27	4	2	1	CYCLOPHO	2	
87	########	518242g	1	SITTALAG	29	2	1	1	1	1	1	28	1	28	3	1	2	NIL	2	
88	########	562028G	1	SHANKAR	27	2	2	2	1	6	3	24	3	24	4	1	1	SLE SYMPT	2	
89	########	850710F	1	SHIVALI PI	29	2	1	1	1	5	0.01	29	0.01	29	4	1	2	NIL	2	
90	########	557125G	1	SUHAB.C.	18	1	2	2	1	1	0.04	18	0.04	18	1	1	1	TYPHOID 2	2	
91	########	072289G	1	SUCHARIT	21	2	1	2	1	2	3	18	3	18	3	2	2	NIL	2	
92	########	561089G	1	SUKANYA	20	2	1	2	1	7	0.1	19	0.1	20	4	2	2	NIL	2	
93	########	485530F	1	SURYA	22	2	1	2	1	1	1	21	1	21	4	2	2	NIL	2	
94	########	368990G	1	SWEETY KI	24	2	1	2	1	1	4	20	1	23	4	2	2	NIL	2	

78	########	7246948	1	SARBANI (	36	2	1	1	1	5	18	18	17	19	4	3	1	RAINY SEA	1	
79	########	171547C	1	SUPRAVA	33	2	1	1	1	7	5	28	5	28	4	1	1	SLE SYMPT	2	
80	########	812750G	1	SIVAKUMA	22	2	1	1	1	3	1	21	0.02	22	3	2	2	NIL	2	
81	########	800461G	1	SHAILYA K	34	2	1	2	1	8	0.08	33	1	33	3	2	2	NIL	2	
82	########	273899D	1	SHIPRA RA	49	3	1	1	1	2	0.06	49	1	48	3	3	1	FEVER	1	
83	########	805027C	1	SHRABAN	27	2	1	1	1	2	11	16	3	25	3	2	2	NIL	2	
85	########	303827C	1	SONALI DI	30	2	1	1	1	2	13	17	1	29	1	1	1	WITH SLE	2	
86	########	732930G	1	SUNAINA	27	2	1	2	1	1	12	15	0.08	27	4	2	1	CYCLOPHO	2	
87	########	518242g	1	SITTALAG	29	2	1	1	1	1	1	28	1	28	3	1	2	NIL	2	
88	########	562028G	1	SHANKAR	27	2	2	2	1	6	3	24	3	24	4	1	1	SLE SYMPT	2	
89	########	850710F	1	SHIVALI PI	29	2	1	1	1	5	0.01	29	0.01	29	4	1	2	NIL	2	
90	########	557125G	1	SUHAB.C.	18	1	2	2	1	1	0.04	18	0.04	18	1	1	1	TYPHOID 2	2	
91	########	072289G	1	SUCHARIT	21	2	1	2	1	2	3	18	3	18	3	2	2	NIL	2	
92	########	561089G	1	SUKANYA	20	2	1	2	1	7	0.1	19	0.1	20	4	2	2	NIL	2	
93	########	485530F	1	SURYA	22	2	1	2	1	1	1	21	1	21	4	2	2	NIL	2	
94	########	368990G	1	SWEETY KI	24	2	1	2	1	1	4	20	1	23	4	2	2	NIL	2	
95	########	846122G	1	TAPAN CH	43	3	2	1	2	9	2	41	0.09	43	2	2	2	NIL	2	
96	########	853256G	1	TASDER	20	2	2	2	1	2	1	19	0.08	20	3	2	2	NIL	2	
97	########	112912C	1	TERINMAF	45	3	1	1	1	1	16	29	16	29	3	3	1	SURGERY (	2	
98	########	326895G	2	VASAMTH	55	4	1	1	1	3	1	54	25	30	3	2	2	NIL	1	
99	########	724509G	1	VARSHA	32	2	1	1	1	8	2	30	2	30	4	2	2	NIL	2	
100	########	753599D	1	VITHYA	27	2	1	1	1	1	4	23	0.01	27	2	2	2	NIL	2	
101	########	545503A	1	VIJAYALA	41	3	1	1	1	1	1	40	0.05	41	3	2	2	NIL	2	

alopwsym	pandif	exacrbxn	durexrb	imprwme	hairgb	thyroid	cutlesion	skininvl	photo	oralulc	nasalulc	jointp	chestp	dyspnea	abdop	renal	periods	abortion	trim	como
1	2	1	0.01	1	2	2	2	2	1	1	2	1	1	1	1	2	2	1	0	4
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1	2	2	0	1	2	2	3	2	2	2	2	2	1	2	2	1	1	1	1	
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2	2	1	2	1	2	2	3	2	2	1	2	1	2	1	2	1	2	1	1
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1	2	1	0.05	1	2	2	3	2	1	1	2	1	1	1	1	1	2	0	4
2	2	1	0.02	2	2	2	3	2	1	2	1	1	1	2	2	2	1	0	4
1	2	1	12	2	2	1	3	2	2	1	2	1	2	1	2	1	2	0	4
1	2	1	12	2	2	2	3	2	1	1	1	1	2	2	2	2	1	0	4

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1	2	1	6	2	1	2	3	1	2	1	2	1	2	2	2	2	1	0	4
1	2	1	2	1	2	2	3	2	1	1	1	1	2	2	2	2	1	0	4
1	1	2	4	2	2	2	2	2	2	2	2	2	2	2	2	2	4	0	4
1	2	2	0	2	2	2	3	2	2	1	2	1	2	2	2	2	1	0	4
2	2	1	2	2	2	2	2	1	1	1	2	2	2	2	2	2	1	0	4
1	2	2	0	1	2	2	4	2	2	1	2	2	2	2	2	1	2	0	4
1	2	2	0	1	2	2	3	2	1	1	2	1	2	2	2	1	2	0	4
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1	2	2	0	1	2	2	4	3	2	2	2	1	2	2	1	2	1	0	4
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1	2	1	3	1	2	1	3	1	1	1	2	1	2	2	2	1	2	0	4
1	2	2	0	1	2	1	2	1	1	1	2	2	2	2	2	1	1	0	4
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1	2	2	0	1	2	2	2	2	1	1	2	1	1	2	2	1	2	0	4
2	4	2	0	2	2	1	2	2	1	2	2	1	2	2	2	2	1	0	4
2	2	1	0.05	2	2	1	3	1	2	2	2	1	2	2	2	2	1	1	1
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1	2	2	0	1	2	2	3	2	1	2	2	1	2	2	2	2	4	0	4
1	2	2	0	1	2	2	4	3	2	2	2	2	1	1	2	1	1	0	4
1	2	2	0	1	2	2	3	2	1	1	2	1	2	1	2	2	1	2	1
1	2	1	0.02	1	2	2	3	2	2	1	1	1	2	2	2	2	1	0	4
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1	2	1	12	1	2	2	4	3	1	1	1	1	2	2	2	2	1	1	1
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75	1	2	2	0	1	2	2	4	3	2	2	2	2	1	1	2	1	1	0	4	
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96	1	2	1	1	2	2	1	2	2	2	1	1	1	1	1	2	1	1	0	4	
97	1	1	2	0	1	2	2	2	2	2	2	2	1	1	1	1	2	3	0	4	

1	comorbs	dm	htn	tb	ba	otherco	sx6	stress6	illness	dieting	food	shampoo	chemical	straight	famsiz	famalop	dsaffect	change	sunscrn	topster	topci
2	2	2	2	2	2	NIL	2	2	NIL	2	3	3	2	1	2	2	5	4	1	1	
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7	1	2	2	2	2	HYPOTHY	2	2	NIL	2	3	1	2	2	2	2	1	4	1	1	
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9	1	1	2	2	2	PULM EME	2	1	PNEUMOI	2	3	7	2	2	2	2	3	3	2	2	
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1	topci	steroid	adju	antimal	slicc	sledsc	dbp	sbp	pulse	temp	acle	scle	ocle	otherc	nsa	dnsa	pnsa	lupush	sa	mucosa	conj
2	2	1	3	1	1	14	88	110	140	1	2	3	9	NIL	1	1	2	2	2	2	
3	2	2	3	1	1	10	80	120	76	1	1	3	9	NIL	1	1	2	2	2	2	
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6	2	1	10	2	1	2	70	112	76	1	3	3	9	NIL	1	1	1	2	2	2	
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1	fydte	faa	fydaa	fbd	fbh	ffh	fch	feh	fdle	fhc	fhd	fvp	fvd	fsfut	fmt	ftv	fav	tempo	ttaga	thdd	tvelh
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23	3	2	3	2	2	2	2	2	1									2	2	2	

24	3	1	3	1	2	2	2	2	2	177	195.9	14.5	13.3	46.46	0.059	5.9	3.61	2	2	2
25	3	2	3	2	2	2	2	2	2	216	239.1	41.2	75.3	46.61	0.048	1.43	1.95	2	1	1
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37	1	2	3	2	2	2	2	2	2	165	182.7	22.1	21	47.19	0.056	3.52	3.52	2	2	2
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91	3	2	3	2	2	2	2	2	2	155	171.6	49.6	67.5	53.68	0.039	1.02	2.73	2	2	2
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76	3	2	3	2	2	2	2	2	2	288	318.8	22.8	52	39.72	0.051	3.39	1.9	2	2	2
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87	3	2	3	2	2	2	2	2	2	169	187.1	16.3	18.8	52.13	0.065	5.13	2.85	2	2	2
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89	3	2	3	2	2	2	2	2	2	217	240.2	16.3	27.7	48.36	0.047	5.13	3.78	2	1	1
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91	3	2	3	2	2	2	2	2	2	155	171.6	49.6	67.5	53.68	0.039	1.02	2.73	2	2	2
92	3	2	3	2	2	2	2	2	2	157	173.8	63.3	68.6	51.11	0.035	0.58	1.8	2	1	1
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94	3	2	3	2	2	2	2	2	2	239	264.6	15.7	32.1	42.62	0.056	7.47	4.21	2	2	2
95	3	2	3	2	2	2	2	2	2	158	174.9	53.8	69.7	56.99	0.04	0.56	3.33	1	2	2
96	3	2	3	2	2	2	2	2	2	205	226.9	31	49.8	49.19	0.056	2.22	3.83	2	2	2
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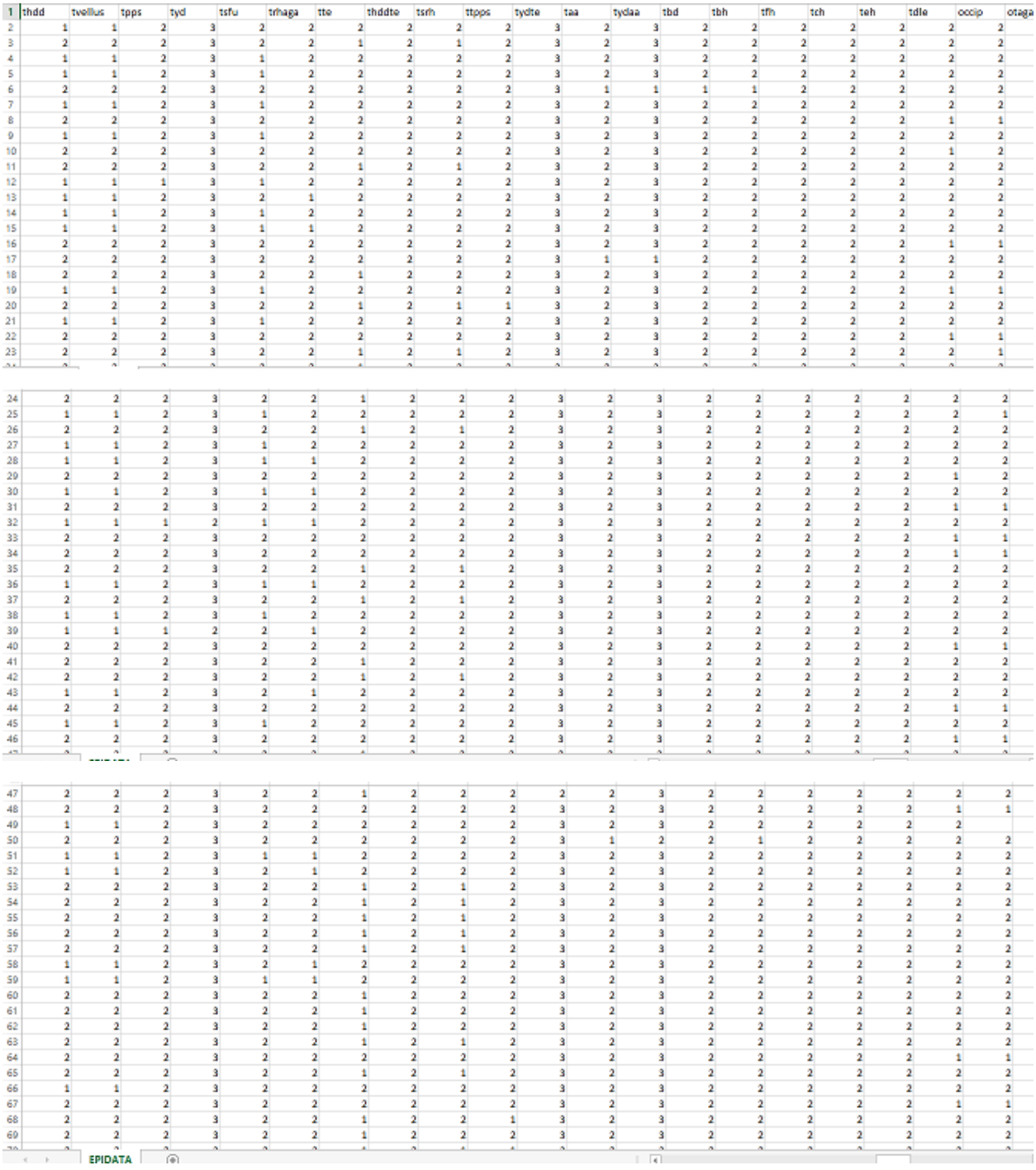

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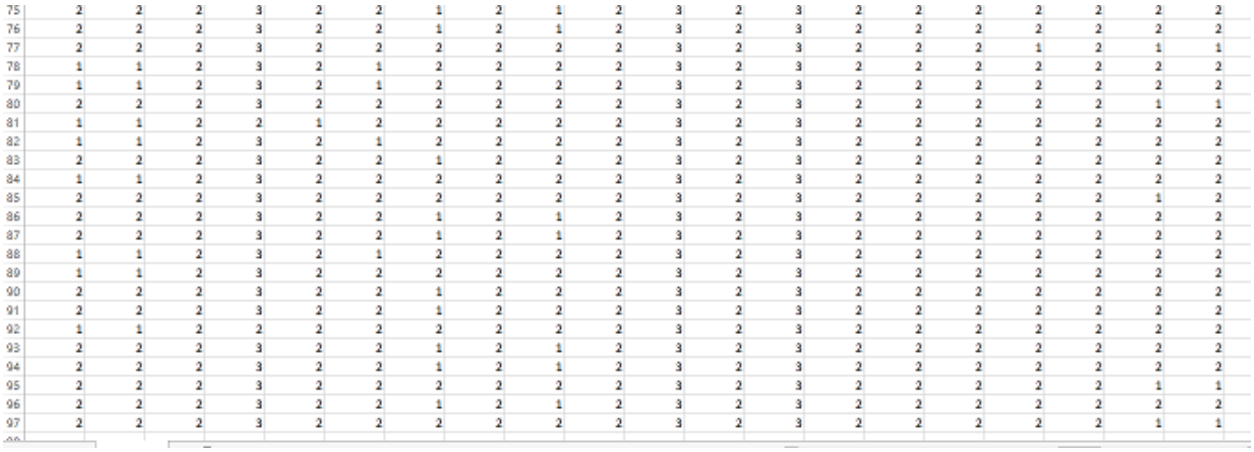

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24	252	279	25	44.3	43.51	0.056	3	2.64	2	2	2	2	2	2	2	2	2	2	3	3
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27	199	220.3	53	58.7	42.31	0.038	0.89	3.54	2	2	2	2	2	2	2	2	2	2	3	3
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31									1	2	1	2	1	2	2	1	1	2	3	3
32	133	147.2	60.4	60.9	65.59	0.039	0.65	0.57	2	2	2	2	2	2	2	2	2	2	3	3
33									1	2	1	1	1	2	1	1	1	1	3	1
34									1	2	1	2	1	2	2	2	1	1	3	2
35	253	280.1	28.9	60.9	42.42	0.052	2.46	2.73	2	2	2	2	2	2	2	2	2	2	3	3
36	272	301.1	25.5	55.4	43.17	0.049	2.92	3.46	2	2	2	2	2	2	2	2	2	2	3	3
37	157	173.8	21	23.2	47.37	0.062	3.76	3.54	2	2	2	2	2	2	2	2	2	2	3	3
38	260	287.8	30.2	67.5	42.96	0.055	2.31	1.59	2	2	2	2	2	2	2	2	2	2	3	3
39	246	272.3	61.6	147.2	35.29	0.037	0.62	2.48	2	2	2	2	2	2	2	2	2	2	3	3
40									1	2	2	1	1	1	2	1	2	2	2	3
41									2	2	2	2	2	2	2	2	2	2	3	3
42	173	191.5	17.3	18.8	48.94	0.016	4.78	7.19	2	2	2	2	2	2	2	2	2	2	3	3
43	211	233.6	13.6	22.1	46.09	0.062	6.35	3.46	2	2	2	2	2	2	2	2	2	2	3	3
44									1	2	1	1	1	1	2	2	1	1	2	3
45	258	285.6	29.8	62	44.44	0.054	2.35	2.48	2	2	2	2	2	2	2	2	2	2	3	3
46									1	1	1	1	1	2	2	1	1	1	3	3
47	139	153.9	21.3	23.3	48.05	0.059	3.73	6.47	2	2	2	2	2	2	2	2	2	2	3	3

48									1	2	2	1	1	1	2	2	1	1	3	3
49									2	2	2	2	2	2	2	2	2	2	3	3
50									2	2	2	2	2	2	2	2	2	2	3	3
51	147	273.4	42.8	78.6	42.98	0.041	1.34	6.19	2	2	2	2	2	2	2	2	2	2	3	3
52	223	246.9	20	41	43.09	0.057	4	5.62	2	2	2	2	2	2	2	2	2	2	3	3
53	229	253.5	33.8	53.1	47.58	0.043	1.96	3.9	2	2	2	2	2	2	2	2	2	2	3	3
54	295	326.6	19.1	49.8	38.1	0.056	4.23	3.37	2	2	2	2	2	2	2	2	2	2	3	3
55	225	249.1	23.8	38.7	50.82	0.05	3.2	2.5	2	2	2	2	2	2	2	2	2	2	3	3
56									2	2	2	2	2	2	2	2	2	2	3	3
57	198	219.2	19.6	32.1	43	0.053	4.1	5.17	2	2	2	2	2	2	2	2	2	2	3	3
58	183	202.6	35.9	50.9	51.85	0.053	1.78	3.56	2	2	2	2	2	2	2	2	2	2	3	3
59	169	187.1	35.2	49.8	48.96	0.044	1.84	1.98	2	2	2	2	2	2	2	2	2	2	3	3
60	250	276.8	21.7	50.9	40.65	0.052	3.61	3.42	2	2	2	2	2	2	2	2	2	2	3	3
61	192	212.6	8.5	12.2	38.14	0.065	10.76	3.58	2	2	2	2	2	2	2	2	2	2	3	3
62	191	211.4	9	13.3	41.84	0.058	10.11	2.62	1	2	2	2	1	2	2	1	2	2	3	3
63	193	213.7	19	28.8	50.93	0.07	4.26	4.95	2	2	2	2	2	2	2	2	2	2	3	3
64									1	2	1	2	1	2	1	1	1	1	3	2
65	246	272.3	32.3	59.8	46.83	0.053	2.09	2.41	2	2	2	2	2	2	2	2	2	2	3	3
66	200	221.4	17.1	27.7	45.87	0.06	4.85	4.4	2	2	2	2	2	2	2	2	2	2	3	3
67									1	2	2	2	1	2	2	2	1	1	3	3
68	158	174.9	20.2	18.8	52.69	0.061	3.95	2.5	2	2	2	2	2	2	2	2	2	2	3	3
69	395	437.3	39.4	143.9	27.42	0.048	1.54	1.64	2	2	2	2	2	2	2	2	2	2	3	3
70	296	327.7	18.9	52	30.43	0.057	4.29	2.19	2	2	2	2	2	2	2	2	2	2	3	3
71									2	2	2	2	2	2	2	2	2	2	3	3

75	149	165	17.2	16.6	58.7	0.063	4.81	5.21	2	2	2	2	2	2	2	2	2	2	3	3
76	266	294.5	14.6	28.8	33.83	0.051	5.85	6.75	2	2	2	2	2	2	2	2	2	2	3	3
77									1	2	2	2	1	2	2	1	2	2	1	3
78	204	225.8	20	32.1	44.55	0.054	4	3.38	2	2	2	2	2	2	2	2	2	2	3	3
79	176	194.8	31.1	42.1	55.24	0.05	2.21	3.12	2	2	2	2	2	2	2	2	2	2	3	3
80									1	2	1	2	1	2	2	1	2	2	3	3
81	262	290	27.2	52	35.61	0.055	2.63	3.11	2	2	2	2	2	2	2	2	2	2	3	3
82	196	217	53.2	65.3	44.86	0.042	0.88	3.83	2	2	2	2	2	2	2	2	2	2	3	3
83	223	246.9	17	31	42.62	0.058	4.88	3.85	2	2	2	2	2	2	2	2	2	2	3	3
84	218	241.3	5.8	11.1	47.86	0.059	16.24	3.38	2	2	2	2	2	2	2	2	2	2	3	3
85	206	228.1	15	25.5	49.59	0.066	5.67	4.1	1	2	1	1	2	1	2	2	1	1	3	3
86	103	114	7.5	4.4	56.72	0.062	12.33	4.88	2	2	2	2	2	2	2	2	2	2	3	3
87	208	230.3	36.9	49.8	41.67	0.05	1.71	3.69	2	2	2	2	2	2	2	2	2	2	3	3
88									2	2	2	2	2	2	2	2	2	2	3	3
89	151	167.2	35.8	32.1	54.44	0.044	1.79	2	2	2	2	2	2	2	2	2	2	2	3	3
90	193	213.7	36.8	58.7	52.83	0.046	1.72	3.24	2	2	2	2	2	2	2	2	2	2	3	3
91	183	202.6	38.3	50.9	47.06	0.044	1.61	2.08	2	2	2	2	2	2	2	2	2	2	3	3
92	155	171.6	52	43.2	50	0.038	0.92	1.88	2	2	2	2	2	2	2	2	2	2	3	3
93	271	300	27.9	56.5	43.66	0.052	2.58	3.46	2	2	2	2	2	2	2	2	2	2	3	3
94	177	195.9	11.8	15.5	45.45	0.063	7.47	4.95	2	2	2	2	2	2	2	2	2	2	3	3
95									1	2	1	1	1	2	1	1	1	1	2	3
96	290	321	28.9	73.1	43.24	0.05	2.46	2.56	2	2	2	2	2	2	2	2	2	2	3	3
97									1	2	1	2	2	2	1	2	1	2	3	3
98																							

	aasalt	alasalt	tsh	fnb	tc	n	e	b	f	m	plt	iron	ferritin	vitb12	vitd	tdxt	tdxt	tdxb	vhpt	vhpb	ciaga
2	0	0	2.28	11.8	3800	53	2	0	33	12	202000	108	102.8	2000	29.8	2	1	2	2		
3	0	0	5.66	11.1	5500	64	1	0	28	7	153000	25	11.1	210	50.3	2	2	2	2	2	
4	0	0	3.26	9.8	13600	73	2	0	17	8	322000	38	109.5	414	21.5	1	1	2	2	2	
5	0	0	1.99	6.7	2600	52	0	0	36	12	48000	60	1073	1860	31.4	2	1	1			
6	100	0	2.82	13.6	11800	85	0	0	6	9	248000	70	31.1	407	32.3	3	3	3			
7	0	0	0.8	11.6	10200	86	0	0	8	6	141000	30	35.2	207	25.9	1	1	2			
8	0	29	0.65	11.9	8100	51	1	0	36	12	209000	110	33.8	418	29.3	4	4	4			
9	0	0	1.51	6.2	18200	76	1	0	17	6	261000	10	1075	436	11.6	1	1	1			
10	0	1	1.2	11.5	5700	78	2	0	11	9	263000	100	8.3	377	20	1	4	1	1	1	
11	0	0	0.91	10.2	5600	82	0	0	15	3	140000	22	18.8	269	29.8	1	2	2			
12	0	0	1.35	11.6	4400	69	2	0	18	11	160000	151	44.1	1397	62	1	1	2			
13	0	0	1.83	11.4	13100	62	0	0	31	7	272000	22	18.7	398	9.5	2	1	2			
14	0	0	0.99	8.7	2500	31	5	0	7	7	314000	46	204	531	20.8	1	1	2			
15	0	0	1.69	9.9	4700	76	0	0	15	9	227000	52	60	465	25.6	2	1	2			
16	0	3	3.31	10.1	5400	72	0	0	19	9	150000			141	53.5	1	4	4			
17	10	0	5.47	10.8	5000	60	1	0	30	7	57000	51	19.9	336	19.9	1	3	3			
18	2	0	1.99	8.2	22300	85	0	0	5	10	244000	56	413.7	2000	7	3	2	2			
19	0	12	2.59	10	6100	76	1	0	18	5	210000	28	7.3	231	30.8	1	1	4			
20	0	0	3.62	12.6	3300	40	0	0	40	20	182000	62	18.7	177	44	2	2	2			
21	0	0	4.74	10.8	7800						193000	39	19.1	278	31.5	1	1	1			
22	0	45	5.36	11.2	5300	47	7	0	38	0	151000	87	40.3	159	19.6	4	4	4			
23	0	1	1.06	16.6	5500	58	2	0	27	13	249000	100	84	349	19.9	4	2	4			

23	0	1	1.06	16.6	5500	58	2	0	27	13	249000	100	84	349	19.9	4	2	4			
24	0	0	3.11	12.2	5200	60	1	0	30	9	271000	72	71.4	269	22.7	3	2	2			
25	0	1	0.65	13.7	8000	33	0	0	15	2	254000					1		4			
26	0	0	0.66	13.2	5300	81	0	0	16	3	228000	70	34.2	292	16.8	2	2	2			
27	0	0	6.41	13	8000	59	2	0	32	7	123000	99	372	359	40.8	1	1	1			
28	0	0	1.8	8.9	6900	85	0	0	8	7	53000	41	113.6	937	30.4	1	1	1			
29	0	19	0.41	10.2	6000	65	4	0	24	7	167000	33	175.8	273	29.8	2	4	4			
30	0	8	2.57	10	5500	42	20	0	31	7	84000	45	24.5	341	16	4	2	2			
31	0	2	3.02	9.5	2000	38	0	0	10	2	152000	65	3060	422	22.4	4	4	4			
32	0	0	0.42	5.4	2600	70	2	0	18	10	56000	27	503.1	2000	6.3	1	1	1			
33	0	32	6.18	10.7	6300	59	3	0	26	12	185000	41	93	230	29.2	4	4	4			
34	0	23	2.97	12.3	5300	67	1	0	18	14	141000	87	626.3	704	70	4	4	4			
35	0	0	4.39	11.7	8100	31	0	8	58	3	267000	49		797	11.2	2	2	2			
36	0	0	0.46	11.7	11700	71	0	0	23	6	377000			1597	38.3	2	1	2			
37	0	0	4.8	8.9	4300	72	0	0	24	4	148000				12.9	2	2	2			
38	0	0	0.66	13.2	1800	86	0	0	7	7	225000	36	3097	2000	49.7	2	1	2			
39	0	0	3.34	8.9	2400	77	0	0	12	11	169000	120	467.9	231	17.5	1	1	1			
40	0	17	0.35	11.8	6800	86	1	0	10	3	294000	76	82.4	476	31.7	4	4	4			
41	0	0	3.98	12.2	3500	71	1	0	17	11	63000	166	58.6	516	20.6	2	2	2			
42	0	0	1.16	11.5	4400	64	0	0	20	8	70000	25		2844	27.4	2	2	2			
43	0	0	1.1	8.3	6100	73	0	0	18	9	171000	11	241.7	285	7.2	2	1	2			
44	0	4	0.04	12.8	3500	38	0	1	52	9	152000	57	161.4	510	20.2	4	4	4			
45	0	0	9.58	10.3	7800	92	0	0	5	3	361000	21	15.8	504	25.5	2	1	2			

47	0	0	1.18	13	10600	85	0	0	13	2	323000			1769	28.8	2	2	2			
48	0	13	0.69	12	5100	60	1	0	23	16	262000	33	14.8	141	27	4	4	4			
49	0	0	0.71	10.8	5800	67	1	0	26	6	111000	23		291	36.2	1	1				
50	17	0	1.21	14.7	7200	63	1	0	28	10	218000					3	3	3			
51	0	0	5.57	10.2	5400	66	1	0	26	7	249000	35	67.5	291	20.5	1	1	1			
52	0	0		12.3	8200	64	1	0	30	5	197000				17.8	1	1	2			
53	0	0	0.2	10.3	11500	53	20	0	21	6	180000	27	942.1	432	15.4	1	2	1			
54	0	0	0.36	11.1	5300	67	1	0	25	7	234000	54	44	260	19	2	2	2			
55	0	0	1.46	9	4100	83	0	0	14	3	206000	81		455	6.6	1	2	1			
56	0	0	2.5	11.8	7500	67	2	0	24	7	258000					2	2	2			
57	0	0	3.11	12.1	13100	59	0	0	35	6	351000					2	2	2			
58	0	0	0.72	7.8	15200	91	0	0	6	3	299000	43	2.3	415	27.6	2	1	2			
59	0	0	2.81	6.3	3500	78	2	0	16	4	83000	21		1110		2	1	2			
60	0	0	5.05	14.2	6800						280000	49	61.6	556	17.9	1	2	2			
61	0	0	1.25	10.7	9100	93	0	0	2	5	370000	23	412.8	1474	7.9	2	2	2			
62	0	3	3.46	9.2	7300	51	1	0	40	8	258000	30	105.6	345	36.1	4	2	2			
63	0	0	1.01	7.3	10900	57	2	0	35	6	71000			336	29.5	2	2	2			
64	0	44	0.83	9.6	4100	82	0	0	16	2	134000	76	367.2	279	30.9	4	4	4			
65	0	0	0.65	9	6300	80	0	0	12	7	395000	11	186.7	244	19.8	2	2	1			
66	0	0	0.7	12	6100	47	2	0	39	12	105000	19	5	334	20	1	1	2			
67	0	2	1.4	12.5	5700	67	0	0	19	10	203000	80	102.4	188	70	4	4	4			
68	0	0	1.92	14.7	4500	77	0	0	15	8	209000	70	104.3	414	40	2	2	2			
69	0	0	6.56	8.2	4500	65	1	0	28	6	94000	45	254.7	235	30.5	2	2	2			

75	0	0	2.97	9.4	4900	66	1	0	32	0	121000	29		290	14.4	2	2	2
76	0	0	3.78	11.1	12000	79	0	0	11	10	231000			253	15.5	2	2	2
77	0	7		9	6900						16100	82		445	28.6	4	4	4
78	0	0	5.16	10.2	9300	90	0	0	7	3	191000			927	70	2	1	2
79	0	0	4.58	10.9	3900	46	1	1	40	12	64000					2	1	2
80	0	6	0.78	9.5	8800	79	0	0	12	9	76000	76	33.7	392	26.8	4	4	4
81	0	0	2.7	11.8	6800	59	1	0	31	9	117000	35	134.8		12.8	2	1	1
82	0	0	1.14	6.9	2600	43	0	0	6	1	110000	33	220.7	1088	18.3	1	1	1
83	0	0	0.19	13.5	3700	51	4	0	32	13	210000	22	422.1	168	13.6	2	2	2
84	0	0	1.32	9.7	13300	77	0	0	11	12	181000	16	577.8	682	7.8	1	1	1
85	0	2	3.82	10.6	8800	75	0	0	21	1	198000	44	188.4	412	11.9	4	4	2
86	0	0	4.11	9.4	5700	79	2	0	15	4	309000	29	506.8	388	13	2	2	2
87	0	0	4.12	14.7	6600	55	0	0	37	8	170000	24	329.4	352	37.3	2	2	2
88	0	0	0.12	6	2400	70	0	0	24	6	58000	56	1199.9	176	26.2	2	2	2
89	0	0	0.86	3	9000	52	2	2	35	9	155000	282	30.4	2000	40.9	1	1	2
90	0	0	1.09	12.1	8200	70	0	0	27	3	130000	47	87.8	2000	32.3	2	2	2
91	0	0	2.23	13	10100	74	0	0	18	8	260000					1	2	2
92	0	0	4.79	9.2	4500	55	0	0	30	15	171000		1076.4	339	13.7	1	1	1
93	0	0	2.24	11.4	9500	80	0	0	12	8	9500	33		2000	39.4	2	2	2
94	0	0	0.02	13.5	11600	54	1	0	33	12	280000	94	165.1	441	61.9	2	2	2
95	0	10	3.62	10.5	7500	71	0	0	25	4	246000	90	316.9	299	17.1	1	4	4
96	0	0	9.15	12.3	5000	61	1	1	29	8	287000	79		289	40.9	2	2	2
97	0	2	1.25	11.8	3800	54	5	0	31	10	237000	74	87.6	854	28.1	4	4	4

KEY TO THE DATASHEET

SLNO	SERIAL NO	<IDNUM>
DATE	DATE	<dd/mm/yyyy>
CMCHNO	HOSPITAL NUMUBER	_____
INFRMT	INFORMANT?	# (1-SELF, 2-RELATIVE)
NAME	NAME	
_____		
AGE	AGE	##
AG	AGE GROUP	# (1- LESS THAN 20, 2-20-39, 3-40-49, 4-50-59, 5-60-69, 6-70-79, 7- MORE THAN 80)
SEX	SEX	# (1- FEMALE, 2- MALE)
MARSTAT	MARITAL STATUS	# (1-MARRIED, 2-UNMARRIED, 3- SEPARATED, 4- WIDOW)
INDIAN	INDIAN	# (1-INDIAN, 2-NOT INDIAN)
STATE	STATE	# (1-TAMIL NADU, 2-WEST BENGAL, 3-ANDHRA PRADESH, 4-KERELA, 5-BIHAR, 6-JHARKHAND, 7-NORTH EAST INDIA, 8-CHATTISGARH, 9-NOT INDIAN)
DURXN	DURATION OF DISEASE	YEARS ##.## MONTHS
AO	AGE OF ONSET	##
DURALOP	DURATION OF ALOPECIA	YEARS ##.## MONTHS
AOA	AGE OF ONSET OF ALOPECIA	YEARS ##.## MONTHS
AMONTALOP	AMOUNT OF HAIRFALL PER DAY	# (1:0-50, 2:50-100, 3:100-200, 4:>200)
CORSALOP	COURSE OF ALOPECIA	# (1-EPISODIC, 2-CONTINUOUS, 3-INTERMITTENT, 4-NA)
TRIG	ANY TRIGGER?	# (1-YES, 2-NO)
TRIGGER	WHAT TRIGGER	_____
ALOPBDX	HAIRFALL BEFORE DX OF SLE?	# (1- YES, 2-NO)
ALOPWSYM	HAIRFALL WITH OTHER SLE SYMPTOMS	# (1- YES, 2-NO)
PANDIF	PATCHY OR DIFFUSE?	# (1-PATCHY, 2-DIFFUSE, 3-BOTH, 4-NIL)
EXACRBXN	ANY RECENT EXACERBATION	# (1- YES, 2-NO)
DUREXRB	DURATION OF EXACERBATION?	MONTHS ##.## WEEKS
IMPRWMED	ANY IMPROVEMENT WITH MEDICATIONS	# (1- YES, 2-NO)
HAIRGB	EXCESS HAIRGROWTH OTHER AREAS	# (1- YES, 2-NO)
THYROID	THYROID DISEASE	# (1- YES, 2-NO)

CUTLESION	SCALP OR SKIN LESIONS	# (1- SCALP, 2-SKIN, 3-BOTH, 4-NIL)
SKININVL	SKIN INVOLVEMENT ?	# (1- LOCALISED, 2-GENERALISED, 3-ABSENT)
PHOTO	PHOTOSENSITIVITY	# (1- YES, 2-NO)
ORALULC	ORAL ULCER	# (1- YES, 2-NO)
NASALULC	NASAL ULCER	# (1- YES, 2-NO)
JOINTP	JOINT PAIN	# (1- YES, 2-NO)
CHESTP	CHEST PAIN	# (1- YES, 2-NO)
DYSPNEA	BREATHLESSNESS	# (1- YES, 2-NO)
ABDOP	ABDOMINAL PAIN	# (1- YES, 2-NO)
RENAL	RENAL INVOLVEMENT	# (1- YES, 2-NO)
PERIODS	DO YOU HAVE REGULAR PERIODS?	# (1- YES, 2-NO, 3-POSTMENOPAUSAL, 4-NA)
ABORTION	NUMBER OF ABORTION	##
TRIM	WHICH TRIMESTER?	# (1- FIRST, 2-SECOND, 3-THIRD, 4-NA)
COMORBS	COMORBIDITIES	# (1-PRESENT, 2- ABSENT)
DM	DIABETES	# (1-YES, 2-NO)
HTN	HYPERTENSION	# (1-YES, 2-NO)
TB	TUBERCULOSIS	# (1-YES, 2-NO)
BA	BRONCHIAL ASTHMA	# (1-YES, 2-NO)
OTHERCO	OTHER COMORBIDITIES	_____
SX6	ANY SURGERY IN LAST 6 MONTHS	# (1- YES, 2-NO)
STRESS6	ANY STRESS IN LAST 6 MONTHS	# (1- YES, 2-NO)
ILLNESS	ANY ILLNESS	_____
DIETING	ANY SPECIAL DIETING	# (1- YES, 2-NO)
FOOD	FOOD HABITS	# (1- NVEG, 2-VEG, 3-MIXED)
SHAMPOO	SHAMPOO/WASH IN 1 WEEK	#
CHEMICAL	CHEMICALLY PROCESSED	# (1- YES, 2-NO)
STRAIGHT	HAIR STRAIGHTENING	# (1- YES, 2-NO)
FAMSLE	ANY FAMILY MEMBER WITH SLE	# (1- YES, 2-NO)
FAMALOP	ANY FAMILY MEMBER WITH ALOP	# (1- YES, 2-NO)
DSAFFECT	WAY DISEASE AFFECT YOUR LIFE	# (1- DAILY WORK, 2-JOB, 3-STUDIES, 4-MARRIAGE, 5-NIL)
CHANGE	YOU HAD TO CHANGE/STOP	# (1- CHANGE JOB, 2-STOPPED WORKING, 3- STOPPED STUDYING, 4-NIL)
SUNSCRN	SUNSCREEN?	# (1-YES, 2-NO)
TOPSTER	TOPICAL STEROID	# (1-YES, 2-NO)
TOPCI	TOPICAL CALCINEURIN	# (1-YES, 2-NO)
STEROID	WHICH STEROID?	# (1-PREDNISOLONE, 2-DEFLAZACORT, 3-DEXAMETHASONE, 4-HYDROCORTISONE, 5-METHYLPREDNISOLONE, 6-BETAMETHASONE, 7-NIL)
ADJU	WHICH ADJUVANT?	## (1-MMF, 2-AZATHIOPRINE, 3-METHOTREXATE, 4-CYCLOPHOSPHAMIDE, 5-CICLOSPORINE, 6-DAPSONE, 7-ACITRETIN, 8-IVIG, 9-OTHERS, 10-NIL)
ANTIMAL	WHICH ANTIMALARIAL	## (1-HCQ, 2-CQ, 3-QUINACRINE, 4-NIL)
SLICC	SLICC CRITERIA	# (1- FULFILLED, 2-NOT FULFILLED)
SLEDSC	SLEDAI SCORING	###

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DBP                    DIASTOLIC BP                              ###
SBP                    SYSTOLIC BP                               ###
PULSE                  PULSE/MINUTE                              ###
TEMP                   TEMPERATURE                               # (1- AFEBRILE, 2-
FEBRILE)

ACLE                   ACUTE CUTANEOUS LE                        # (1- MALAR RASH, 2-
GENERALISED RASH, 3-NIL)
SCLE                   SUBACUTE CUTANEOUS LE                     # (1- ANNULAR, 2-
PAPULOSQUAMOUS, 3-NIL)
CCLE                   CHRONIC CUTANEOUS LE                      # (1- CLASSICAL DISCOID,
2-HYPERTROPHIC, 3-LUPUS PROFUNDUS, 4- LUPUS PANNICULITIS, 5- MUCOSAL DLE, 6- LUPUS
TUMIDUS, 7-CHILBLAIN, 8-LICHENOID DLE, 9-NIL)
OTHERC                 OTHER CUTANEOUS
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NSA                    NON SCARRING ALOPECIA                     # (1- PRESENT, 2-ABSENT)
DNSA                   DIFFUSE NON-SCARRING ALOPECIA             # (1- PRESENT, 2-ABSENT)
PNSA                   PATCHY NON-SCARRING ALOPECIA              # (1- PRESENT, 2-ABSENT)
LUPUSH                 LUPUS HAIR                                # (1- PRESENT, 2-ABSENT)
SA                     SCARRING ALOPCIA                          # (1- PRESENT, 2-ABSENT)

MUCOSA                 MUCOSAL ULCER                             # (1- PRESENT, 2-ABSENT)
CONJ                   CONJUNCTIVAL CONGESTION                   # (1- PRESENT, 2-ABSENT)
NASALU                 NASAL ULCER                               # (1- PRESENT, 2-ABSENT)
ORALU                  ORAL ULCER                                # (1- PRESENT, 2-ABSENT)

NAIL                   NAIL CHANGES                              # (1- PRESENT, 2-ABSENT)
PARONY                 PARONYCHIA                                # (1- PRESENT, 2-ABSENT)
PERIUN                 PERIUNGUAL ERYTHEMA                       # (1- PRESENT, 2-ABSENT)

JOINTS                 JOINT CHANGES                             # (1- PRESENT, 2-ABSENT)

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TSCOPY                 TRICHOSCOPIC FINDINGS                     # (1- ABNORMAL, 2-
NORMAL)


FRONT                  FRONTAL SCALP                          #(1-SCARRING, 2-NONSCARRING,
3-BOTH, 4-NIL)

FTAGA                  FRONT AGA PATTERN                         #(1- PRESENT, 2- ABSENT)
FHDD                    HAIR DIAMETER DIVERSITY                  #(1- MORE THAN 20
PERCENT, 2- LESS THAN 20 PERCENT)
FVELLUS                 SHORT VELLUS HAIR                         #1- MORE THAN 10
PERCENT, 2- LESS THAN 10 PERCENT)
FPPS                    PERIPILAR SIGN IN AGA                    #(1- PRESENT, 2- ABSENT)
FYD                     YELLOW DOTS IN AGA                      #(1- NUMEROUS, 2- SCANTY,
3-ABSENT)
FSFU                    PREDOMINANT SINGLE FOLLICULAR UNITS      #(1- PRESENT, 2-ABSENT)
FRHAGA                  SHORT REGROWING HAIR IN AGA              #(1- PRESENT, 2-ABSENT)

FTTE                   FRONTAL TE PATTERN                        #(1- PRESENT, 2- ABSENT)
FHDDTE                  HDD IN TE                                #(1- PRESENT, 2- ABSENT)
FSRH                    SHORT REGROWING HAIR IN TE               #(1- PRESENT, 2-ABSENT)
FTPPS                   PERIPILAR SIGN IN TE                     #(1- PRESENT, 2-ABSENT)
FYDTE                   YELLOW DOTS IN TE          #(1- NUMEROUS, 2- SCANTY, 3-ABSENT)
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FAA                    FRONTAL ALOPECIA AREATA                #(1- PRESENT, 2- ABSENT)
FYDAA                   YELLOW DOTS IN AA                     #(1- NUMEROUS, 2-SCANTY,
3-ABSENT)
FBD                     BLACK DOTS                            #(1- PRESENT, 2-ABSENT)
FBH                     BROKEN HAIRS                          #(1- PRESENT, 2-ABSENT)
FFH                     FLAME HAIR                            #(1- PRESENT, 2-ABSENT)
FCH                     CIRCLE HAIR                           #(1- PRESENT, 2-ABSENT)
FEH                     EXCLAIMATION MARK HAIR                #(1- PRESENT, 2-ABSENT)


FDLE                    FRONTAL DLE                           #(1- PRESENT, 2-ABSENT)


FHC                    FRONTAL HAIR COUNT                     ###
FHD                    FRONTAL HAIR DENSITY                   ###.#
FVP                    FRONTAL VELLUS PERCENTAGE              ##.#
FVD                    FRONTAL VELLUS DENSITY                 ###.#
FSFU                   FRONTAL SINGLE FOLLICULAR UNITS        ##.##
FMT                    FRONTAL MEDIAN THICKNESS               #.###
FTV                    FRONTAL TERMINAL VELLUS RATIO          ##.##
FAV                    FRONTAL ANAGEN TELOGEN RATIO           ##.##

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TEMPO                   TEMPORAL SCALP                       # (1- SCARRING, 2-
NONSCARRING, 3-BOTH, 4-NIL)

TTAGA                   TEMPORAL AGA PATTERN                 #(1- PRESENT, 2- ABSENT)
THDD                     HAIR DIAMETER DIVERSITY             #(1- MORE THAN 20
PERCENT, 2- LESS THAN 20 PERCENT)
TVELLUS                  SHORT VELLUS HAIR                   #(1- MORE THAN 10
PERCENT, 2- LESS THAN 10 PERCENT)
TPPS                     PERIPILAR SIGN IN AGA               # (1- PRESENT, 2- ABSENT)
TYD                      YELLOW DOTS IN AGA                  # (1- NUMEROUS, 2-
SCANTY, 3-ABSENT)
TSFU                     PREDOMINANT SINGLE FOLLICULAR UNITS  # (1- PRESENT, 2-ABSENT)
TRHAGA                   SHORT REGROWING HAIR IN AGA         # (1- PRESENT, 2-ABSENT)

TTE                     TEMPORAL TE PATTERN                  # (1- PRESENT, 2- ABSENT)
THDDTE                   HDD IN TE                           # (1- PRESENT, 2- ABSENT)
TSRH                     SHORT REGROWING HAIR                # (1- PRESENT, 2-ABSENT)
TTPPS                    PERIPILAR SIGN IN TE                # (1- PRESENT, 2-ABSENT)
TYDTE                    YELLOW DOTS IN TE                   # (1- NUMEROUS, 2-
SCANTY, 3-ABSENT)

TAA                     TEMPORAL ALOPECIA AREATA             # (1- PRESENT, 2- ABSENT)
TYDAA                    YELLOW DOTS IN AA                   # (1- NUMEROUS, 2-SCANTY,
3-ABSENT)
TBD                      BLACK DOTS                          # (1- PRESENT, 2-ABSENT)
TBH                      BROKEN HAIRS                        # (1- PRESENT, 2-ABSENT)
TFH                      FLAME HAIR                          # (1- PRESENT, 2-ABSENT)
TCH                      CIRCLE HAIR                         # (1- PRESENT, 2-ABSENT)
TEH                      EXCLAIMATION MARK HAIR              # (1- PRESENT, 2-ABSENT)


TDLE                    TEMPORAL DLE                         # (1- PRESENT, 2-ABSENT)

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OCCIP                  OCCIPITAL SCALP                        # (1- SCARRING, 2-
NONSCARRING, 3-BOTH, 4-NIL)

OTAGA                  OCCIPITAL AGA PATTERN                  # (1- PRESENT, 2-
ABSENT)
OHDD                    HAIR DIAMETER DIVERSITY               # (1- MORE THAN 20
PERCENT, 2- LESS THAN 20 PERCENT)
OVELLUS                 SHORT VELLUS HAIR                     # (1- MORE THAN 10
PERCENT, 2- LESS THAN 10 PERCENT)
OPPS                    PERIPILAR SIGN IN AGA                 # (1- PRESENT, 2-
ABSENT)
OYD                     YELLOW DOTS IN AGA                    # (1- NUMEROUS, 2-
SCANTY, 3-ABSENT)
OSFU                    PREDOMINANT SINGLE FOLLICULAR UNITS   # (1- PRESENT, 2-
ABSENT)
0RHAGA                  SHORT REGROWING HAIR IN AGA           # (1- PRESENT, 2-
ABSENT)

OTE                    OCCIPITAL TE PATTERN                 # (1- PRESENT, 2- ABSENT)
OHDDTE                  HDD IN TE                           # (1- PRESENT, 2- ABSENT)
OSRH                    SHORT REGROWING HAIR                # (1- PRESENT, 2-ABSENT)
OTPPS                   PERIPILAR SIGN IN TE                # (1- PRESENT, 2-ABSENT)
OYDTE                   YELLOW DOTS IN TE                   # (1- NUMEROUS, 2- SCANTY,
3-ABSENT)


OAA                    OCCIPITAL ALOPECIA AREATA             # (1- PRESENT, 2-
ABSENT)
OYDAA                   YELLOW DOTS IN AA                     # (1- NUMEROUS, 2-
SCANTY, 3-ABSENT)
OBD                     BLACK DOTS                          # (1- PRESENT, 2-ABSENT)
OBH                     BROKEN HAIRS                        # (1- PRESENT, 2-ABSENT)
OFH                     FLAME HAIR                          # (1- PRESENT, 2-ABSENT)
OCH                     CIRCLE HAIR                         # (1- PRESENT, 2-ABSENT)
OEH                     EXCLAIMATION MARK HAIR              # (1- PRESENT, 2-ABSENT)


ODLE                   OCCIPITAL DLE                        # (1- PRESENT, 2-ABSENT)


OHC                   OCCIPITAL HAIR COUNT                    ###
OHD                   OCCIPITAL HAIR DENSITY                  ###.#
OVP                   OCCIPITAL VELLUS PERCENTAGE             ##.#
OVD                   OCCIPITAL VELLUS DENSITY                ###.#
OSFU                  OCCIPITAL SINGLE FOLLICULAR UNITS       ##.##
OMT                   OCCIPITAL MEDIAN THICKNESS              #.###
OTV                   OCCIPITAL TERMINAL VELLUS RATIO         ##.##
OAV                   OCCIPITAL ANAGEN TELOGEN RATIO          ##.##

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RDLE                   REPRESENTATIVE DLE                    # (1- PRESENT, 2-ABSENT)

PWH                    PERIFOLLICULAR WHITE HALO             # (1- PRESENT, 2-ABSENT)
HK                     HYPERKERATOTIC PLUGS                  # (1- PRESENT, 2-ABSENT)
TELAN                  TELANGIECTASIAS                       # (1- PRESENT, 2-ABSENT)
SWA                    STRUCTURELESS WHITE AREAS             # (1- PRESENT, 2-ABSENT)
RDG                    RED DOTS OR GLOBULES                  # (1- PRESENT, 2-ABSENT)
SCALE                  SCALING                               # (1- PRESENT, 2-ABSENT)
EHCP                   EXAGGERATED HONEYCOMB PIGMENT NETWORK  # (1- PRESENT, 2-ABSENT)
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BGG                    BLUE GREY DOTS OR GLOBULES             # (1- PRESENT, 2-ABSENT)
SP                     BLUE/GREY/BROWN SPECKED PIGMENTATION   # (1- PRESENT, 2-ABSENT)
SYD                    SMALL YELLOW DOTS                      # (1- NUMEROUS, 2-
SCANTY, 3-ABSENT)
LYD                    LARGE YELLOW DOTS                      # (1- NUMEROUS, 2-
SCANTY, 3-ABSENT)
MRA                    MILKY RED AREAS                        # (1- PRESENT, 2-ABSENT)
LOFU                   LOSS OF FOLLICULAR UNITS               # (1- PRESENT, 2-ABSENT)


AASALT                  SALT AREA SCORE FOR AA                  ###.#
DLESALT                 SALT AREA SCORE FOR DLE                ###.#

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TSH                    TSH                                    ###.##
HB                     HB                                     ##.#
TC                     TOTAL COUNT                            #####
N                      NEUTROPHIL                             ##
E                      EOSINOPHIL                             ##
B                      BASOPHIL                               ##
L                      LYMPHOCYTE                             ##
M                      MONOCYTE                               ##
PLT                    PLATELET                               ######
IRON                   IRON                                   ###
FERRITIN               FERRITIN                               ####.##
VITB12                 VITB12                                 ####
VITD                   VITD                                   ##.#
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TDX                    TRICHOSCOPIC DIAGNOSIS
TDXF                   TRICHOSCOPIC DIAGNOSIS FRONTAL         # (1-AGA, 2- TE, 3-AA,
4-DLE, 5-NORMAL)
TDXT                   TRICHOSCOPIC DIAGNOSIS TEMPORAL        # (1-AGA, 2- TE, 3-AA,
4-DLE, 5-NORMAL)
TDX0                   TRICHOSCOPIC DIAGNOSIS OCCIPITAL       # (1-AGA, 2- TE, 3-AA,
4-DLE, 5-NORMAL)
VHPF                    VELLLUS HAIR > 10%                  # (1- PRESENT, 2- ABSENT)
VHPO                    VELLLUS HAIR > 10%                  # (1- PRESENT, 2- ABSENT)
CLAGAF                  CLINICAL FAGA                       # (1- PRESENT, 2- ABSENT)



TFDD                   FINAL DX FOR DIFFUSE NON-SCARRING    # (1-AGA, 2- TE, 3-AGA +
TE, 4- ABSENT)
TFDAA                  FINAL DX FOR PATCHY AA               # (1-PRESENT, 2- ABSENT)
TFDDLE                 FINAL DX FOR DLE                     # (1-PRESENT, 2- ABSENT)


CLEDX                  CUTANEOUS DIAGNOSIS                    # (1-ACLE, 2-SCLE, 3-
CCLE, 4-ACLE AND SCLE, 5-SCLE AND CCLE, 6-ACLE AND CCLE, 7-NORMAL)
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